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JUNE 1954

THURSTON SCOTT WELTON, M.D., EDITOR

Differential Sacral Neurotomy

Venous Thrombosis and Thromboembolism

Index Issue Vol. 87

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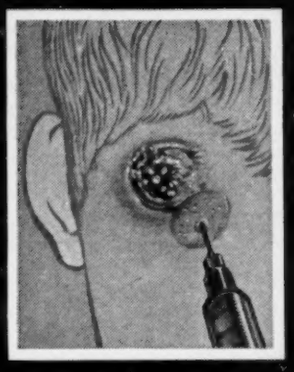

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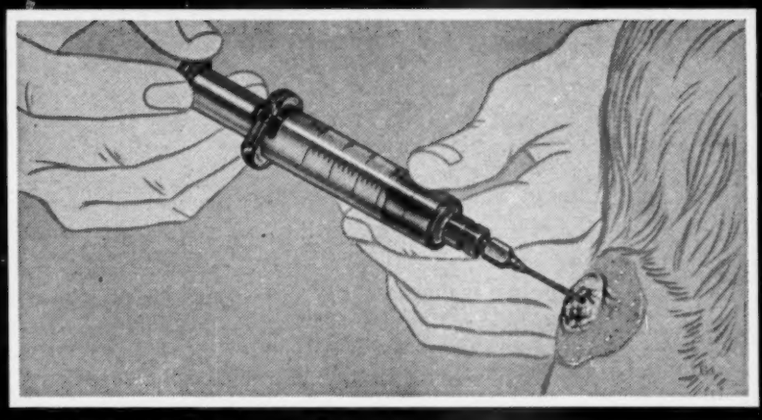

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Clinical Hypoxia 803

PALUEL J. FLAGG

SCIENTIFIC PAPERS

Further Experiments on Animals and Man Supporting the Bioelectrical Method of Measuring the Rate of Healing of Wounds 805

T. CUNLIFFE BARNES AND MARIE D. AMOROSO

Objectives for this research were: investigation of the great variation occurring in measurement of wound potentials, comparison of muscle and skin wounds in animals, studies of reversal of potential occurring at healing and death of the animal, measurements of the local effects of external factors, the specific action of chlorophyll on the rate of healing of experimental human wounds, and studies of the electrical potential of a clinical wound.

Importance of Anatomic Precision in Differential Sacral Neurotomy for "Cord Bladder" 819

J. M. MEREDITH

The author concludes that differential sacral neurotomy is a very satisfactory procedure for achieving an automatic bladder in spastic lesions of the spinal cord either post-traumatically or after non-traumatic cord lesions. Attention is called to certain important anatomic facts and their bearing on surgical technic.

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WILLIAM R. CHAMBERS

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Experiences with the Use of 10 Per Cent Aqueous Phenol for Chemical Sympathectomy. Preliminary Report 830

EARL MILES AND J. S. ROTHMAN

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ALLEN KLEIMAN

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BENEDICT R. HARROW

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LEO H. POLLOCK

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THEODORE D. STEVENSON

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W. N. KEMP

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JULIUS W. TAYLOR

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JULIAN A. STERLING, TIONG-GIOK ONG, IRVING G. KLAUS AND RALPH GOLDSMITH

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JOHN R. SAMPEY

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MAJ. HENRY P. ROSACK, COL. S. W. FRENCH, III AND LT. COL. J. S. PAUL

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WALTER J. PUDERBACH

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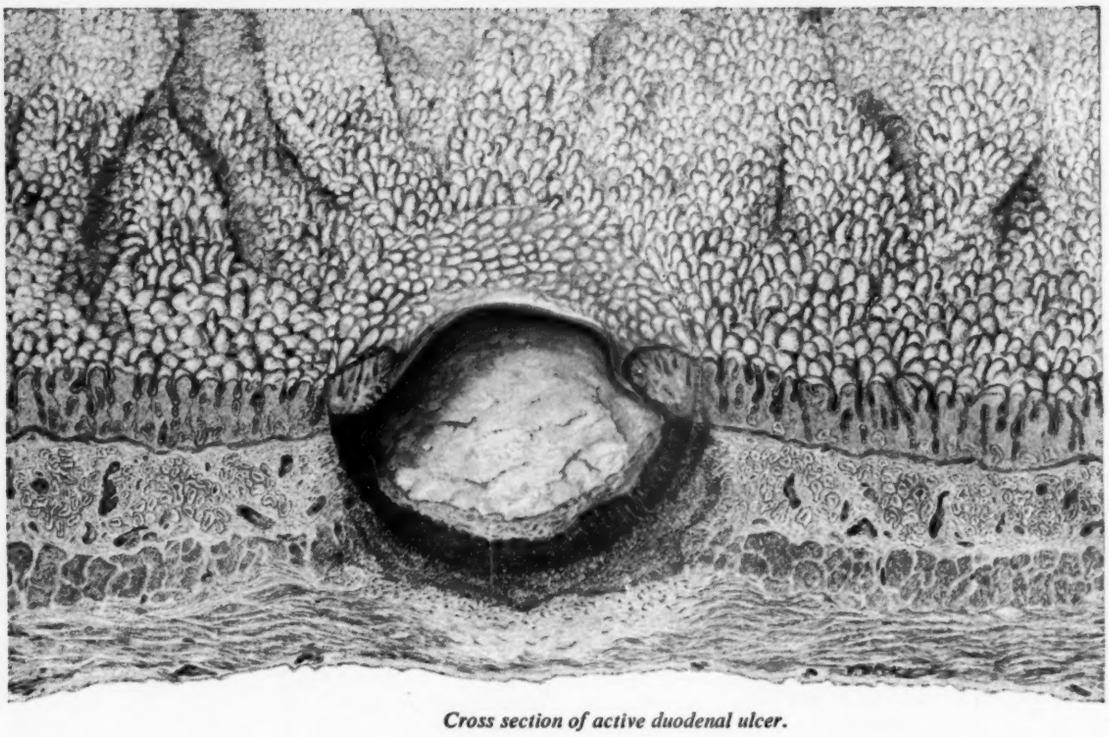

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1. Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.
2. Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthine, Gastroenterology *25*:416 (Nov.) 1953.

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PAUL E. GUTMAN

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MORRIS JOSEPH

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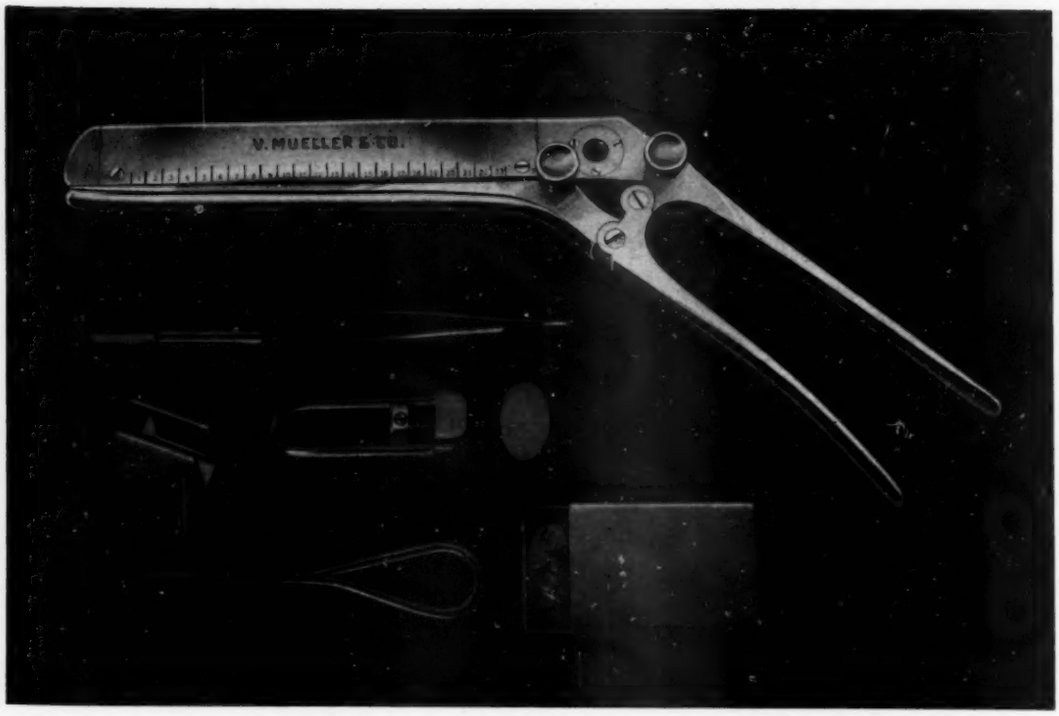

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ROBERT N. WHITTENBERGER

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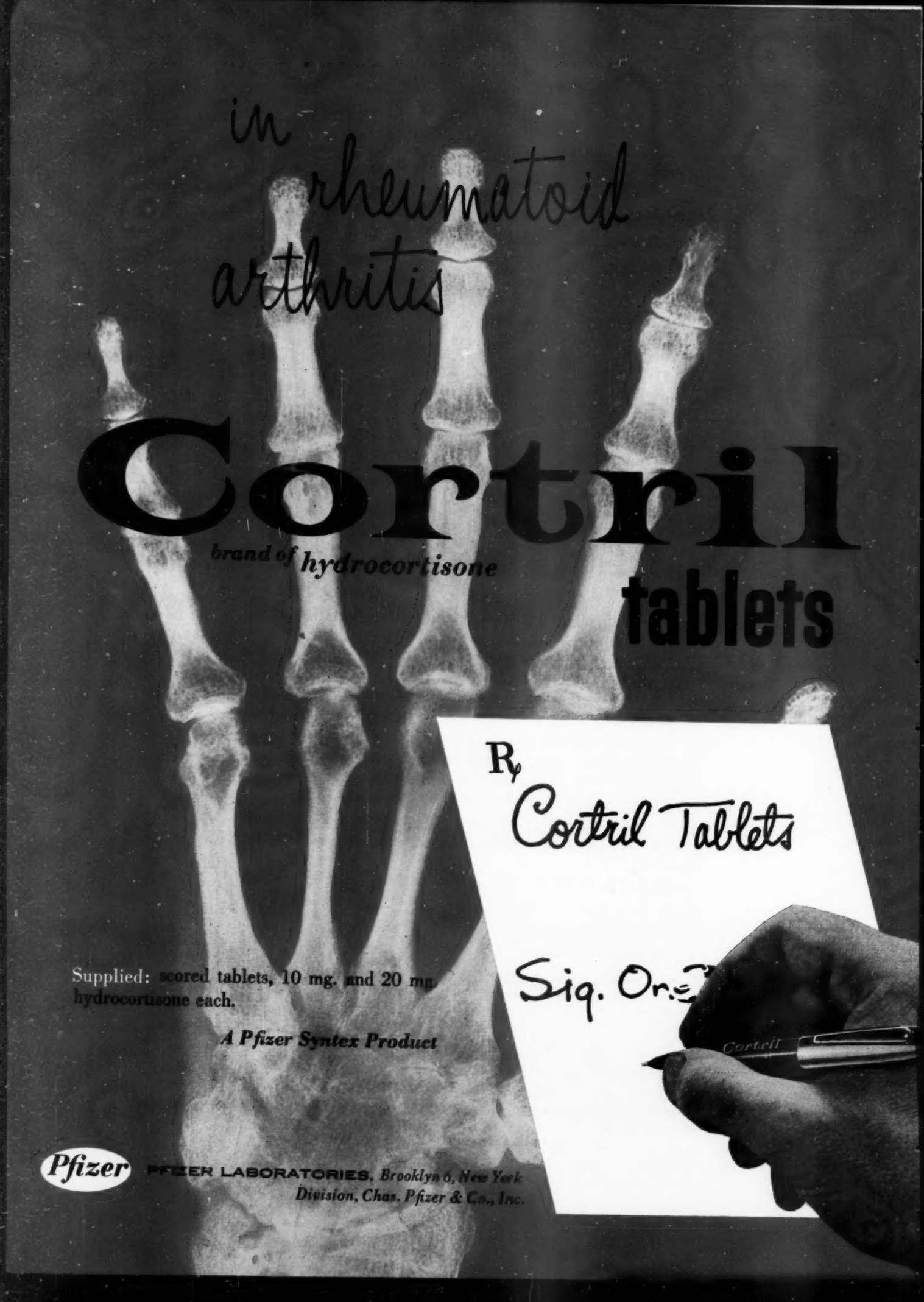

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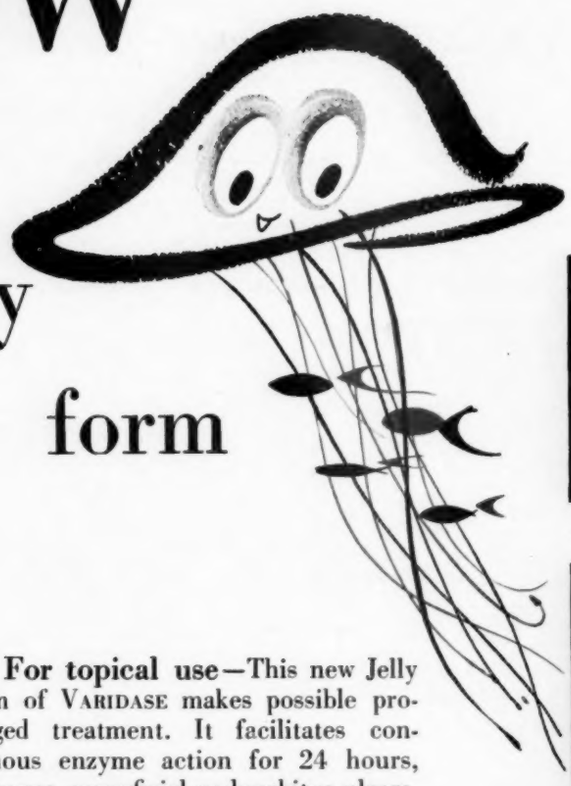

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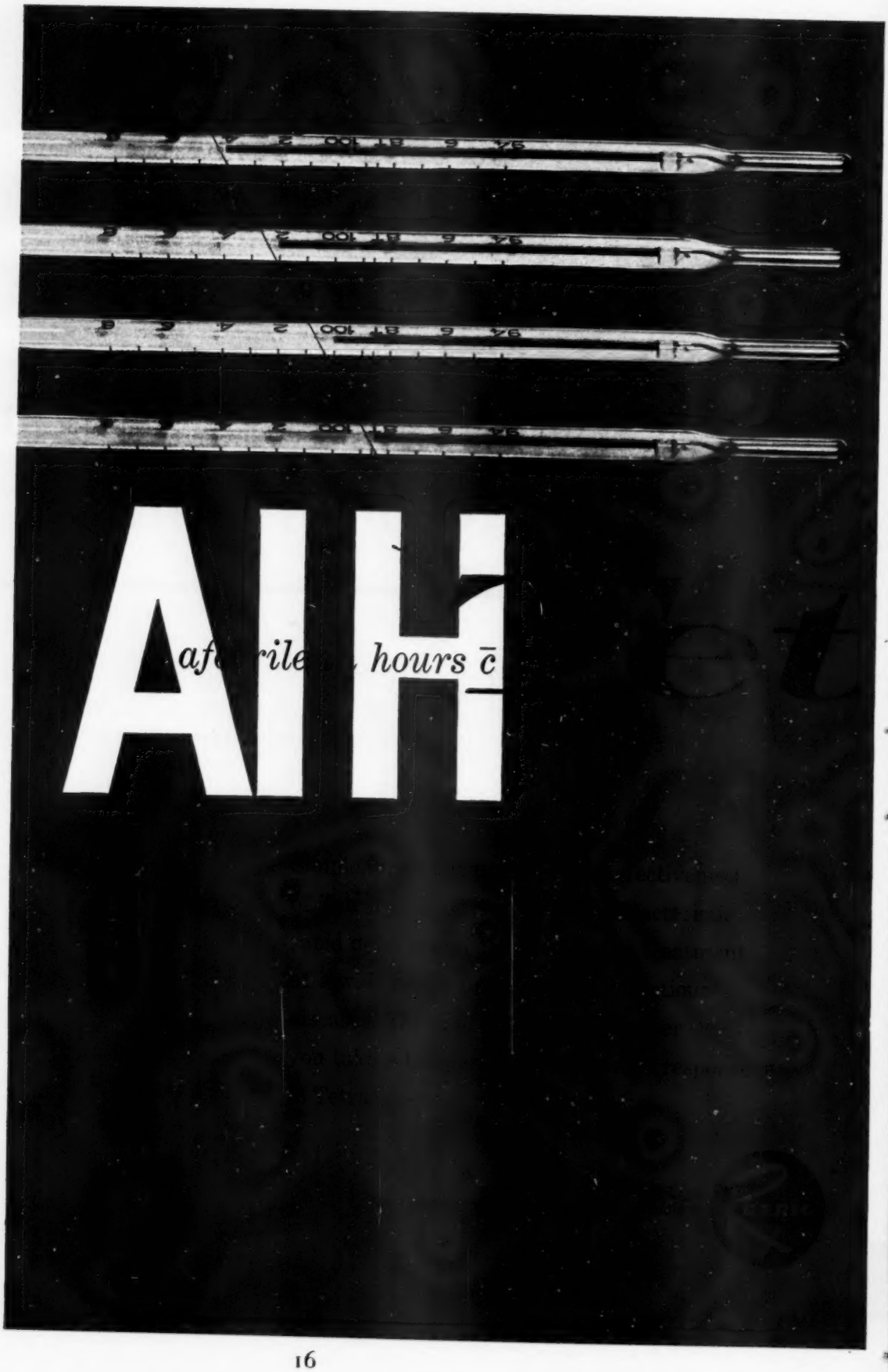
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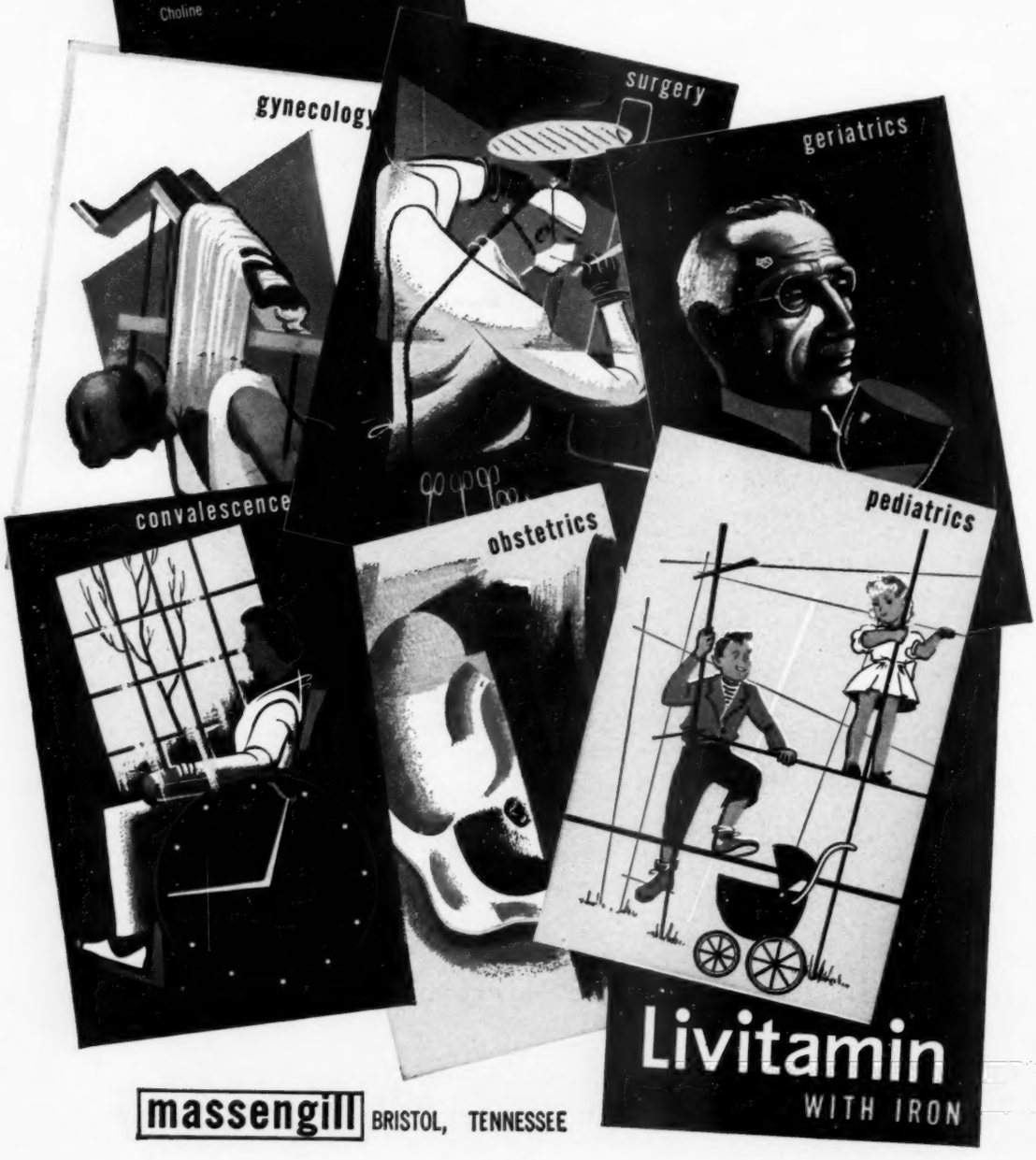

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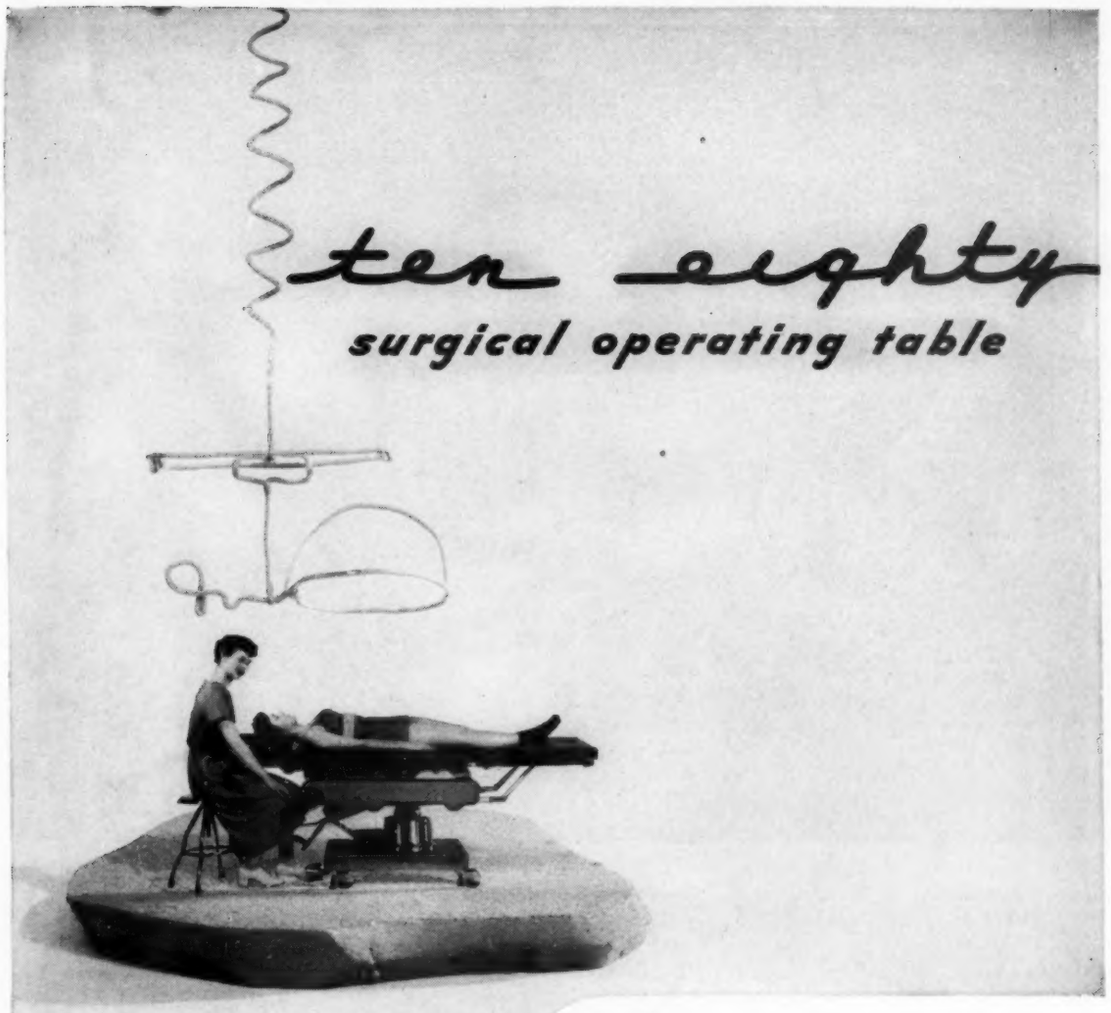

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Menadione (vitamin K analog)	2 mg.

Dosage: 2 capsules daily in severe pathologic conditions; 1 capsule daily when convalescence is established.

Supplied: bottles of 30 and 100.

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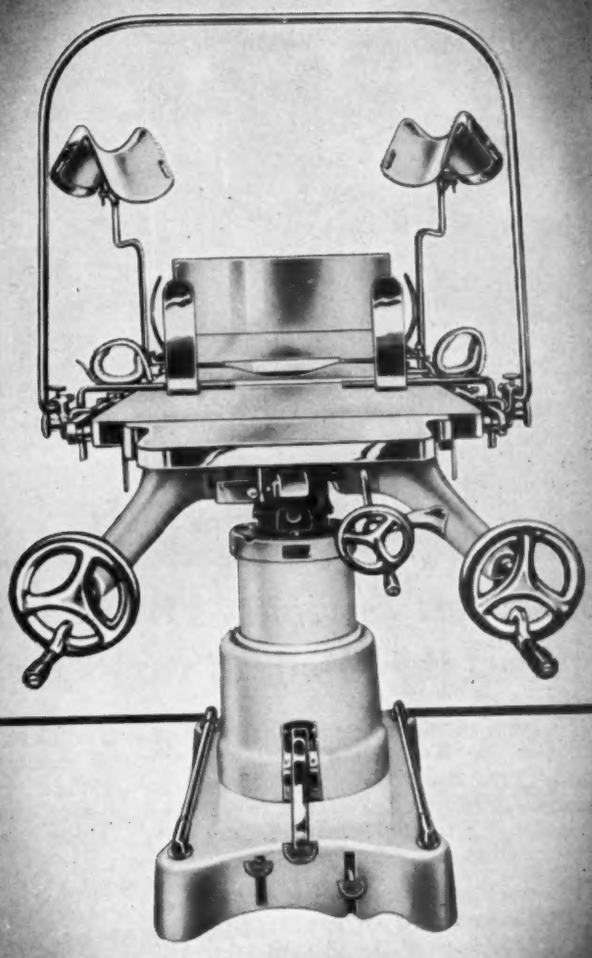
TRUE HEAD-END CONTROL

the Shampaine S-1502



- all controls face anesthetist
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Full versatility of positioning is accomplished by the seated anesthetist.

The surgical team is never disturbed, and personnel is cut to a minimum.

Compare . . . and you'll choose Shampaine!

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These Weck craftsmen represent a combined experience of 122 years in the repair of surgical instruments.

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These men repair surgical instruments in the Weck plant. They are merely typical of their co-workers who constitute America's largest staff of specialists in the repair of Surgical Instruments.

At the service of these men is the most modern repair equipment. But that is only part of the picture—the other part is found in the skill, knowledge and experience of these men who truly deserve the title of "craftsmen".

They know *how* to repair surgical instruments because they know how to *make* surgical instruments and are familiar with the specific uses to which they are put.

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Manufacturers of Surgical Instruments • Hospital Supplies • Instrument Repairing

"For want of a nail the shoe was lost; for want of a shoe the horse was lost; for want of a horse the rider was lost."
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WITH ABBOTT'S revolutionary new Blood Recipient Set, you're doubly protected against blood clogging. The set's cannula has a finely-machined pre-strainer which offers the first barrier to fibrin or clots. A second barrier is provided by the Monel metal filter screen. If the cannula or filter screen clog, simply close the pinch clamp and squeeze the plastic filter chamber several times . . . There's no need to dismantle and reassemble equipment, no need to disturb the patient, no need for a second venipuncture. The Blood Recipient Set is completely disposable. It comes in easy-to-store packages and is sterile, pyrogen-free and ready-to-use. Have your Abbott representative demonstrate it. Or write us direct: Abbott Laboratories, North Chicago, Illinois. Abbott

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Abbott's COMPLETE I.V. AND BLOOD TRANSFUSING Equipment

Sterile, pyrogen-free and ready-to-use

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For Vacuum Collection:

ABBO-VAC®—A-C-D Solution, U.S.P. (N.I.H. Formula B), in universal bottles, 500- and 250-cc. sizes. Blood is drawn directly into container by vacuum.
Available with sterile, disposable Blood Donor Set.

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Abbott A-C-D Blood container—A-C-D Solution, U.S.P. (N.I.H. Formula B), in the familiar Abbo-Liter® intravenous bottles, 500- and 250-cc. sizes. Blood is drawn (closed technique) directly into container by gravity. Available with Sodium Citrate 3% Solution in 500-cc. size. Also available with Donopak 24 and 48, with or without disposable needles. Designed for exclusive use with Abbott i.v. equipment.

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Evacuated Empty Plasma Containers—Sterile evacuated 500- and 250-cc. Universal bottles for storing, transporting and administering plasma or serum.

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Blood Recipient Set—Sterile, disposable, ready-to-use plug-in set for administering blood from any Universal bottle or Abbo-Liter type bottle. Has flexible plastic filter chamber.

VENOPAK®—Abbott's sterile, disposable venoclysis unit for the administration of all intravenous solutions. Converts readily to a blood recipient set with a special, disposable blood filter. For use exclusively with Abbo-Liter® containers.

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Secondary Recipient Set—A unique, disposable unit with a built-in, flexible drip chamber and filter. Designed to plug into any Universal blood bottle and to connect with Abbott's VENOPAK dispensing cap. Allows changeover from saline to blood in a matter of moments, without removing needle from vein.

Secondary VENOPAK—Disposable unit designed for the continuous administration of fluids in the series hookup with VENOPAK.

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SUB-Q-PAK®—A completely disposable, preassembled hypodermoclysis unit with plastic Y tube for administration of fluids subcutaneously.

ADMINISTERING PENTOTHAL® SODIUM

VENOTUBE®—Length of plastic tubing with attached male and female Luer adapters and pinch clamp. Allows anesthesiologist to keep syringe off the patient's arm. Pinch clamp offers additional factor of safety.

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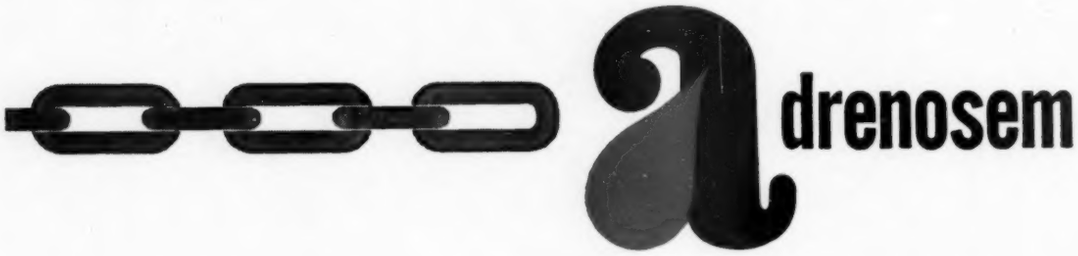
"where oozing from a vascular bed is anticipated"*

ADRENOSEM stops capillary bleeding and oozing. Adrenosem acts directly on the capillary walls to increase resistance and decrease permeability.

There are no reported toxic effects or contraindications attributable to Adrenosem. Adrenosem does not affect blood components or induce embolus formation. It has no sympathomimetic or vasoconstrictive action. Adrenosem is compatible with vitamin K, heparin, and commonly used anesthetics. (It is suggested, however, that antihistamines be withheld for 48 hours prior to and during Adrenosem therapy since antihistamines tend to inactivate Adrenosem.)

*Sherber, D. A.: The Control of Bleeding. Am. J. Surg. *86*:331 (Sept.) 1953.



the missing link in the control of bleeding

The therapeutic regimen for Adrenosem is *simple* and *safe:*

- *preoperatively* to control oozing during surgery and provide clearer operative field: one to two ampuls (5 to 10 mg.) every two hours prior to surgery for two doses.
- *postoperatively* to prevent hemorrhage and check oozing: one to two ampuls (5 to 10 mg.) every two hours until there is no indication of undue bleeding. To maintain control, one ampul may be administered every three hours or 1 to 5 mg. orally t. i. d.
- to control *active bleeding:* One ampul (5 mg. every two hours until bleeding is controlled; frequency of dose may then be diminished.
- to control *severe bleeding:* one ampul (5 mg.) every hour for three doses; then every three hours until bleeding is controlled.
- to control *mild, low-grade bleeding:* 1 ampul (5 mg.) every three or four hours until bleeding is controlled; then 1 to 5 mg. orally four to five times daily until bleeding ceases.
- *maintenance dosage* to prevent bleeding in conditions where small vessel integrity may be impaired: 1 to 5 mg. orally t. i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally every three or four hours; if bleeding persists, oral dosage should be supplemented with 1 ampul (5 mg.) daily.
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Available as Ampuls: 5 mg., 1 cc. (package of 5).
Tablets: 1 mg. S. C. Orange, bottles of 50.
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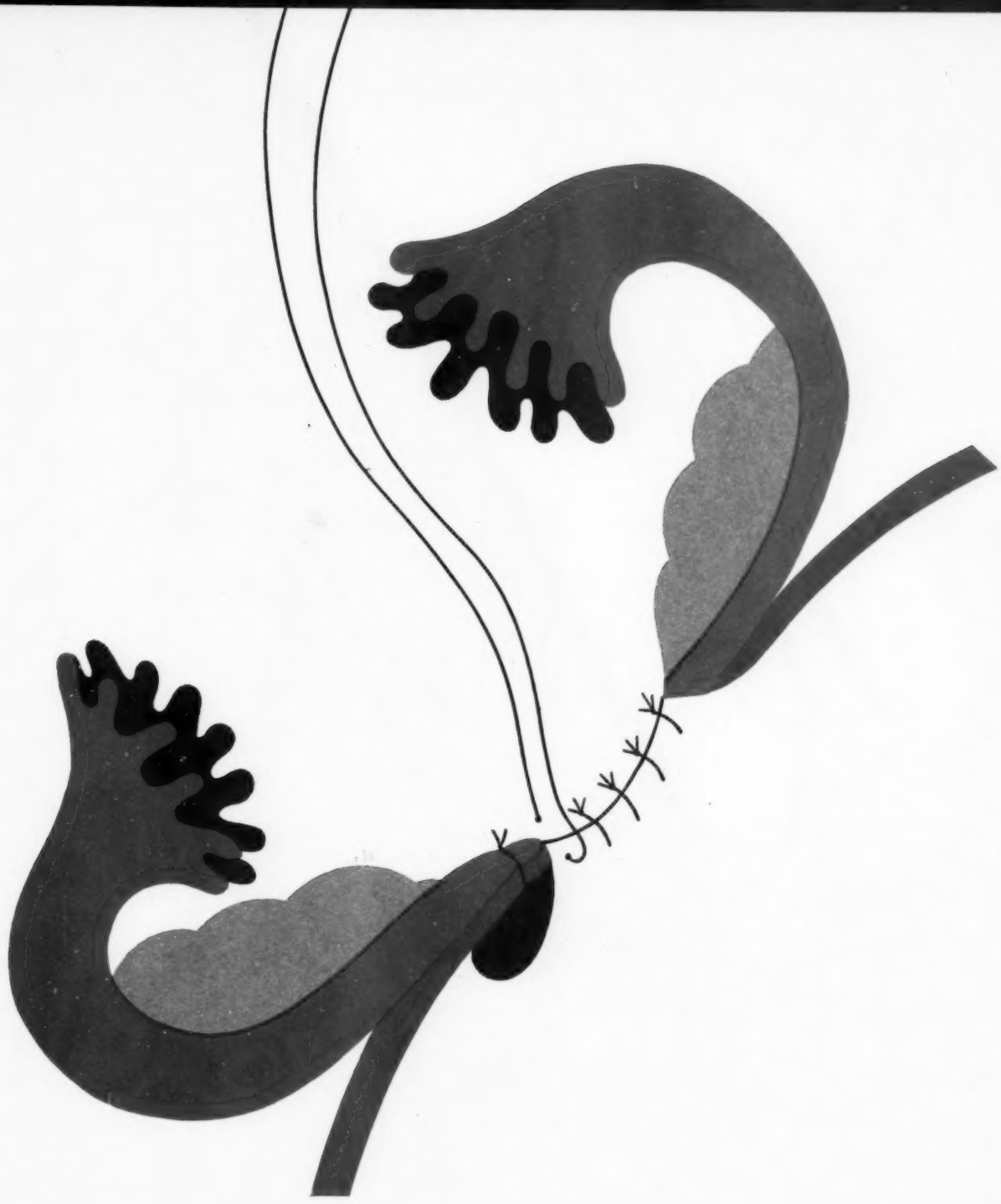

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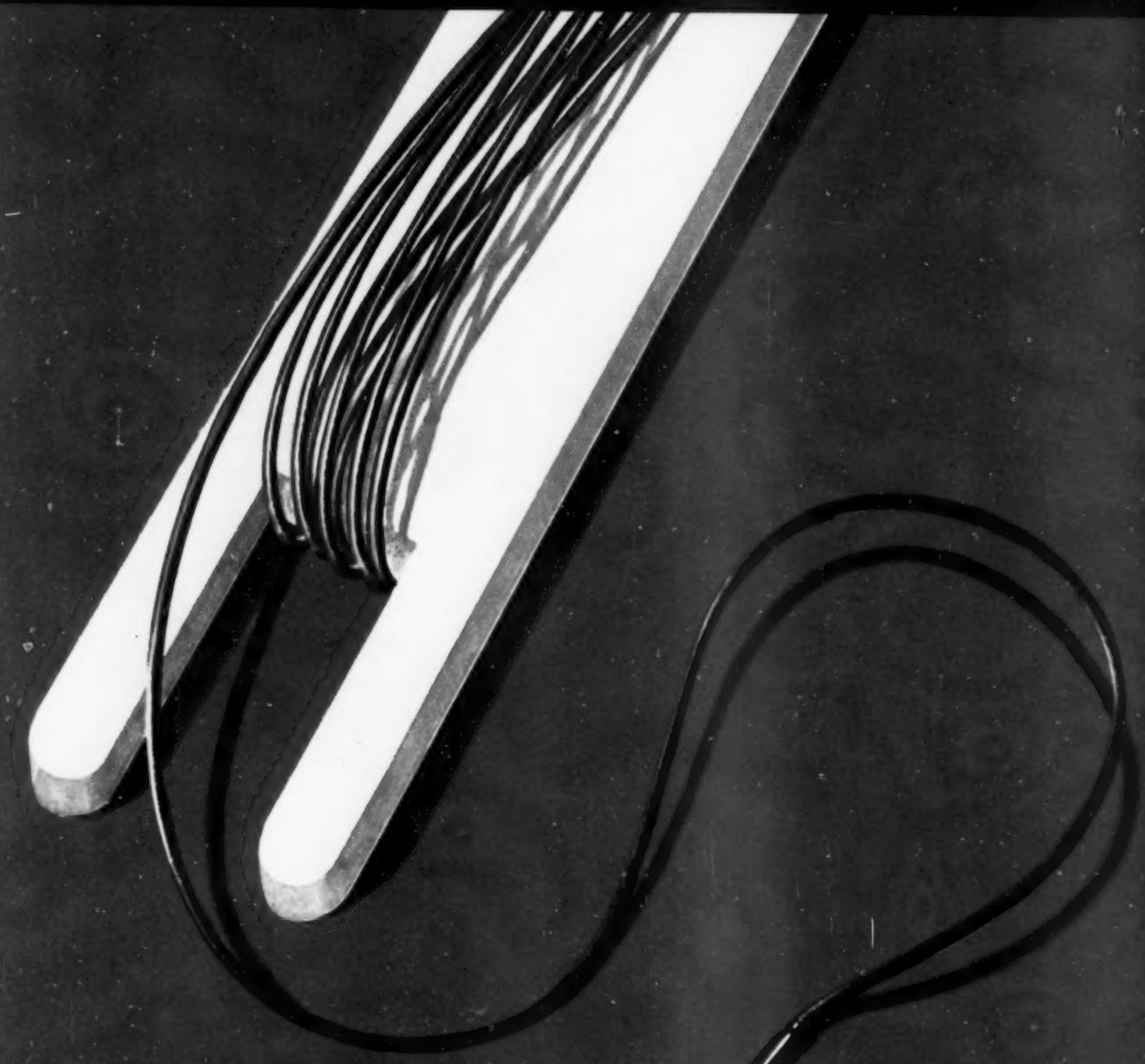

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ATRALOC®

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reduce operating time

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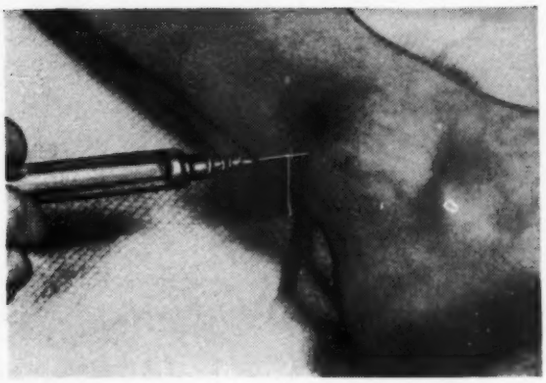

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SPEEDS REDUCTION

- Edema of Accident
- Edema of Surgery (Preoperative Prevention, Postoperative Management)
- Paraphimosis
- Pretibial Swelling in Myxedema
- Hemarthrosis in Hemophilia

Injected directly into the swelling, WYDASE *simplifies* reduction by speeding the intercellular escape of the confined fluid. WYDASE works by enzymatic action—increases the permeability of tissues, accelerates the spread and resorption of trapped transudates and blood. As swellings diminish, so the attendant pain, discoloration, and disability subside. WYDASE—the *modern* approach to the prevention and management of localized edema.

Other uses: WYDASE indications include: increasing the spread of local anesthetics, speeding fluid absorption in hypodermoclysis, combating formation of urinary calculi, facilitating separation of fibrous adhesions.

INJECTION SOLUTION

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HYALURONIDASE (STABILIZED)

Supplied: Vials of 1 cc., 150 TR (turbidity-reducing) units. Also available: Lyophilized WYDASE. *Supplied:* Vials of 150 and 1500 TR units.

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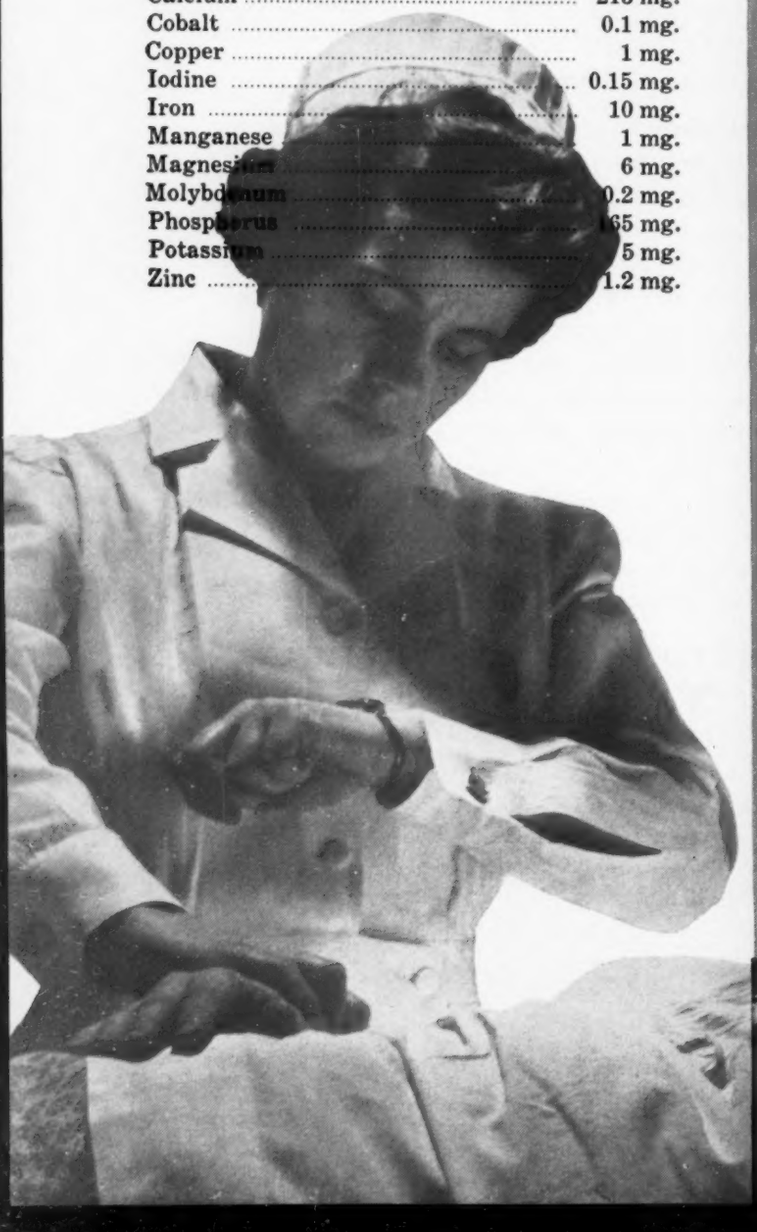
Viterra®

a vitamin-mineral formulation of 21 balanced factors, supplementing the depleted diet

each capsule of *Viterra* contains:

Vitamin A	5,000 U.S.P. Units
Vitamin D	500 U.S.P. Units
Vitamin B_{12}	1 mcg.
Thiamine Hydrochloride	3 mg.
Riboflavin	3 mg.
Pyridoxine Hydrochloride	0.5 mg.
Niacinamide	25 mg.
Ascorbic Acid	50 mg
Calcium Pantothenate	5 mg.
Mixed Tocopherols *(Type IV)*	5 mg.
Calcium	213 mg.
Cobalt	0.1 mg.
Copper	1 mg.
Iodine	0.15 mg.
Iron	10 mg.
Manganese	1 mg.
Magnesium	6 mg.
Molybdenum	0.2 mg.
Phosphorus	165 mg.
Potassium	5 mg.
Zinc	1.2 mg.



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Viterra® Therapeutic

high-potency capsules specifically designed to meet increased nutritional needs during illness

each capsule of

Viterra Therapeutic

contains:

Vitamin A	25,000 U.S.P. Units
Vitamin D	1,000 U.S.P. Units
Thiamine Mononitrate	10 mg.
Riboflavin	5 mg.
Vitamin B_{12}	5 mcg.
Niacinamide	100 mg.
Ascorbic Acid	150 mg.
Calcium	103 mg.
Cobalt	0.1 mg.
Copper	1 mg.
Iodine	0.15 mg.
Iron	10 mg.
Magnesium	6 mg.
Manganese	1 mg.
Molybdenum	0.2 mg.
Phosphorus	80 mg.
Potassium	5 mg.
Zinc	1.2 mg.

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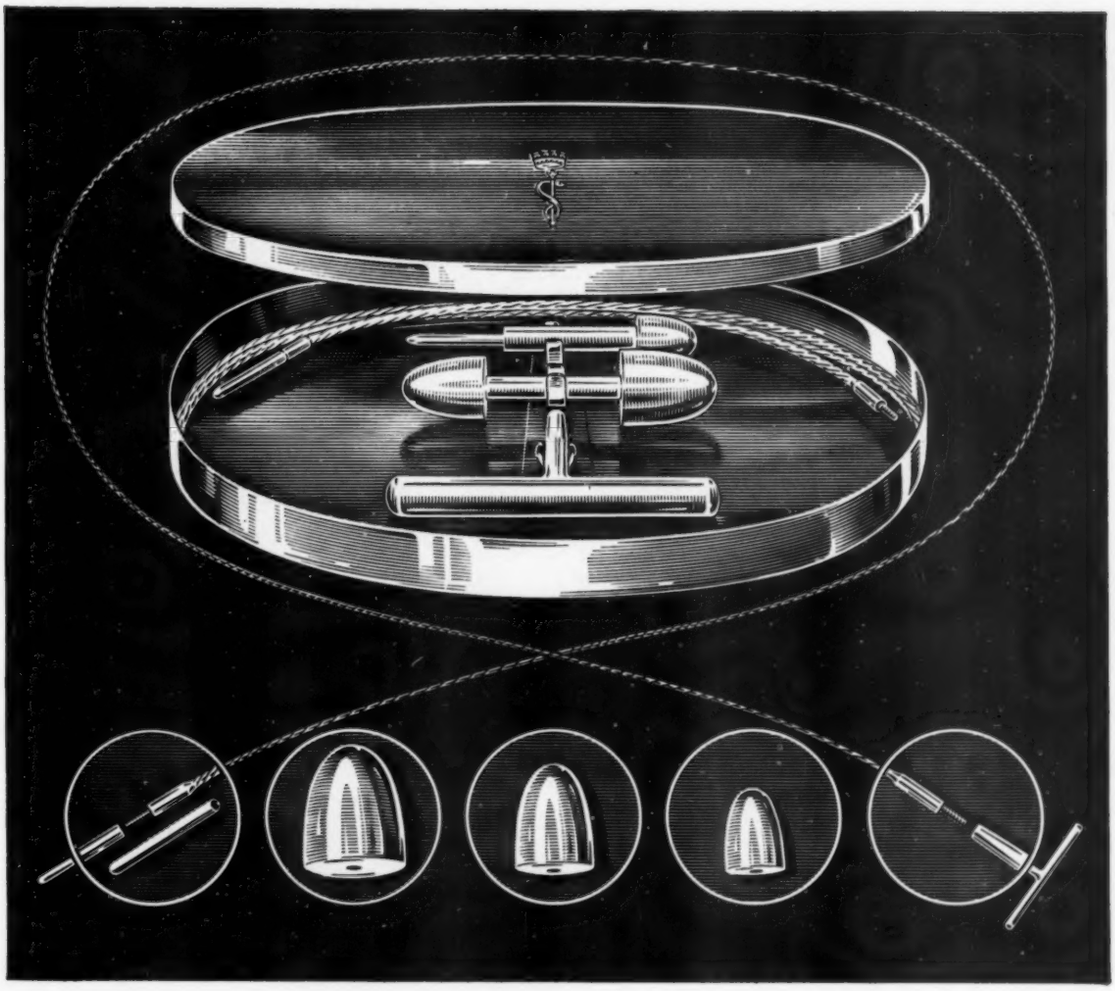

New AN IMPROVED INTRALUMINAL VEIN STRIPPER

DESIGNED BY ROBERT A. NABATOFF, M. D., NEW YORK

Employs a new type of thin, strong, 'twisted-strand' cable. Offers a unique combination of flexibility and firmness. Facilitates guidance of probe tip through even the most tortuous veins.

Cable, 36 inches long, is *threaded on both ends.* Removable handle, probe tip and olives are readily attached to either end.

Removable handle makes stripping easier and surer, minimizes trauma.

Supplied with three olives — 9 mm., 12 mm., 15 mm., two probe tips, one cable and fitted case which permits sterilization as a unit. All parts highly polished, easily cleaned, stainless steel. Repeated sterilization does not affect them.

NOTE:
Ask your surgical dealer to show you this improved vein stripper or write for further information.

Since 1888, surgeons have depended on Kny-Scheerer for the ultimate in instrument craftsmanship. Available exclusively through surgical dealers.

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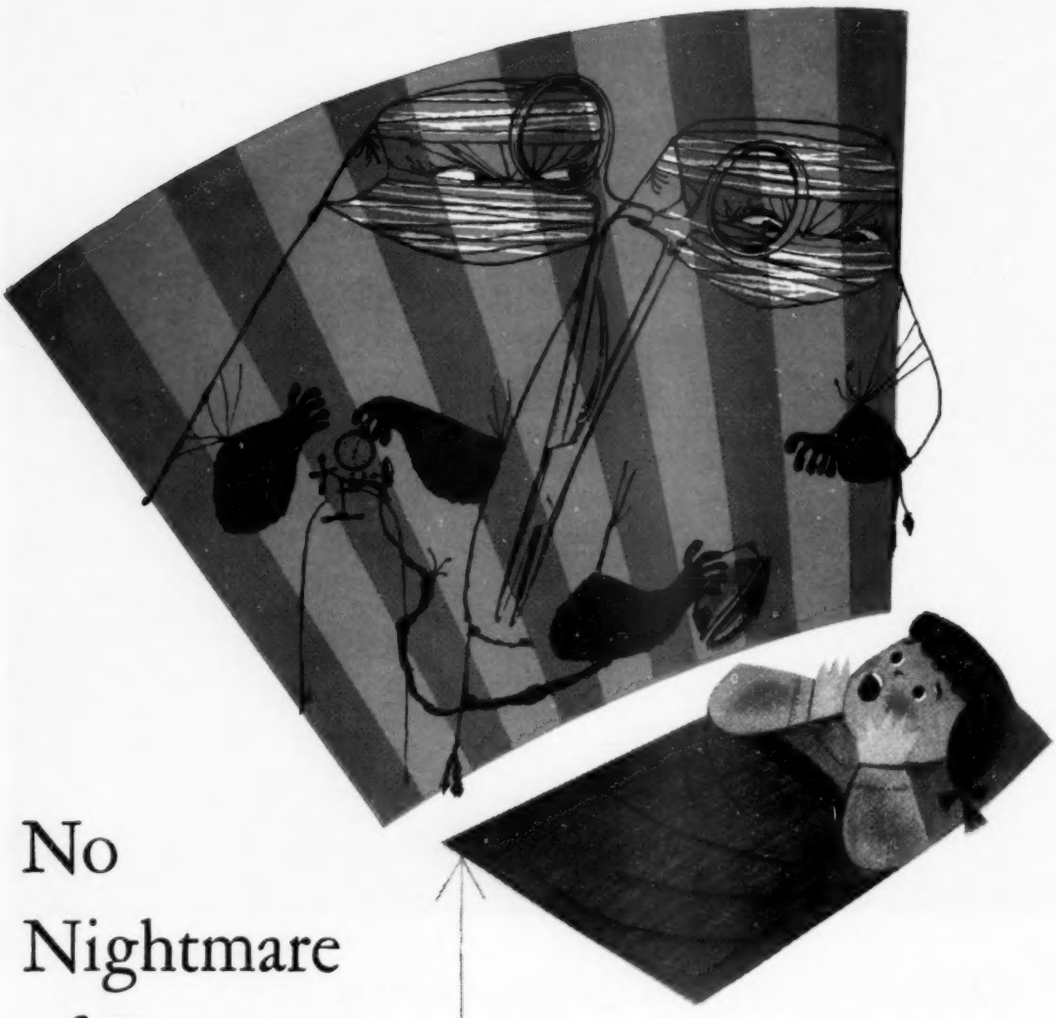

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NO PAIN . . . NO MEMORY

No longer is there a need for the all-too-familiar scene in which a terror-stricken, struggling youngster submits to anesthesia on the operating table. For, with rectal administration of PENTOTHAL® Sodium, the child never knows the operating room. *He falls asleep in his own bed before surgery, awakes there afterward.* PENTOTHAL, in individualized dosages, may be used in a wide variety of circumstances, from preanesthetic sedation to basal anesthesia. With PENTOTHAL, emergence delirium and postanesthetic nausea are minimized. In a number of short, minor surgical procedures, Rectal PENTOTHAL may function as the sole anesthetic agent. Abbott

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"PENTOTHAL Sodium by Rectum," 36-page discussion of PENTOTHAL for preanesthetic hypnosis and basal anesthesia—results from nearly 4,000 cases. Write Abbott Laboratories, North Chicago, Illinois.

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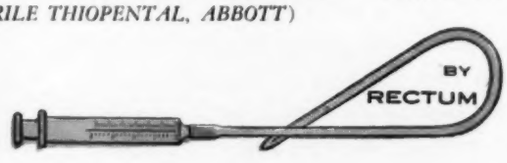
PENTOTHAL® *Sodium*

(STERILE THIOPENTAL, ABBOTT)



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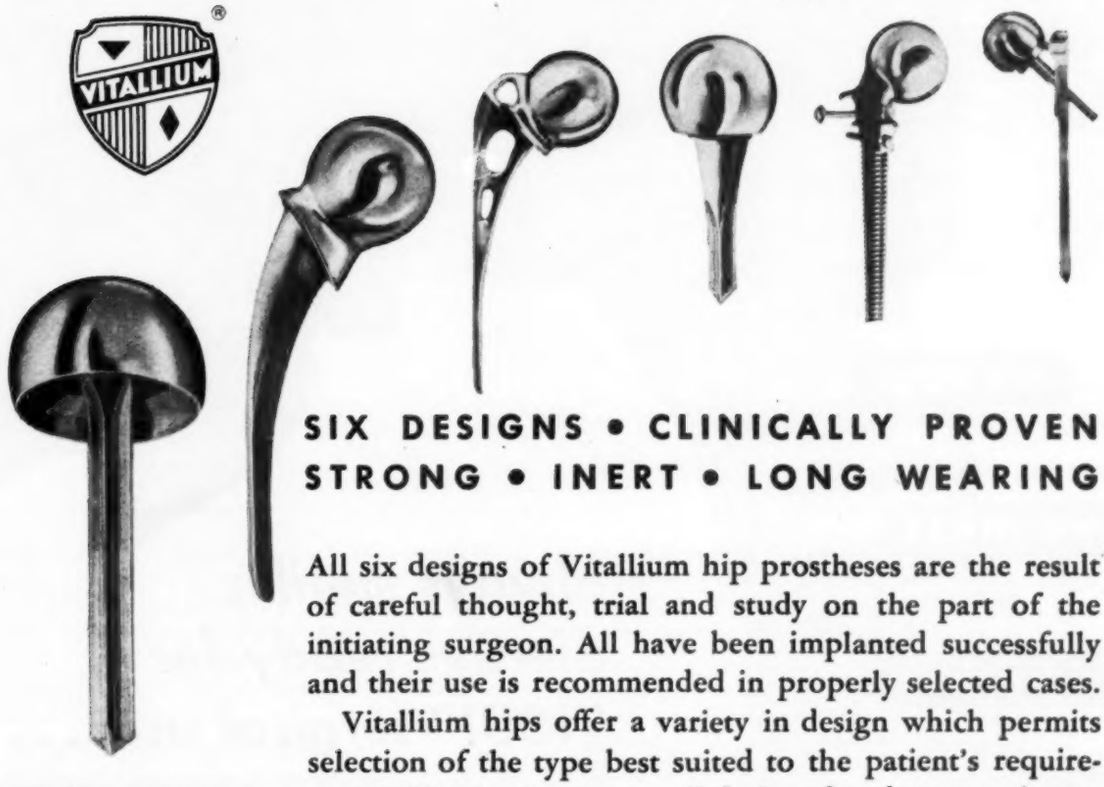
Vitallium

HIP PROSTHESES



SIX DESIGNS • CLINICALLY PROVEN
STRONG • INERT • LONG WEARING

All six designs of Vitallium hip prostheses are the result of careful thought, trial and study on the part of the initiating surgeon. All have been implanted successfully and their use is recommended in properly selected cases.

Vitallium hips offer a variety in design which permits selection of the type best suited to the patient's requirements. They are strong, well-designed and wear-resistant. The head portions are smoothly polished and except for the Lippmann type are hollow for lightness.

Like all Vitallium appliances, the suitability of these prostheses for permanent implantation has been proven throughout 15 years of use.

VITALLIUM HIP DESIGNS
(from left to right)

1. Modified Judet Type No. 6902
2. F. R. Thompson Type No. 6936
3. Moore Type No. 6939
4. J. E. M. Thompson Type No. 6938
5. McBride Type No. 6935
6. Lippmann Type No. 6932

* * *

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Sterile Petrolatum Gauze Dressings

always sterile, always ready for "1001" surgical uses . . .

Outstandingly successful for burns and abrasions, these sterile-packed, ready-made dressings have countless other uses in surgery. Particularly indicated for:

WOUND COVERING, *as for traumatic injuries and after surgery* . . . to protect from irritation and contamination, to avoid adherence.

PACKING, *as in abscess cavity* . . . to permit healing from the bottom, to meet aseptic precautions.

PLUG, *as after hemorrhoidectomy* . . . to help control bleeding without sticking and subsequent tearing.

DRAIN, *as for septic wounds* . . . to avoid maceration, pressure necrosis and erosion.

Available in three sizes:

No. 1—3" x 36" (6 in carton)
No. 2—3" x 18" (12 in carton)
No. 3—6" x 36" (6 in carton)

Obtain from your regular source of supply . . . insist on the ready-to-use, dependably sterile dressings in the foil-envelopes.

CHESEBROUGH MFG. CO., CONS'D

Professional Products Division • **NEW YORK 4, N. Y.**

VASELINE is the registered trade-mark of the Chesebrough Mfg. Co., Cons'd

Meats-in-a-Can

and Kitchen-Cooked Meats...
Comparative Nutritive Values

From a practical dietary standpoint, meats-in-a-can—preserved by commercial canning—are nutritionally interchangeable with meats of like variety prepared in the home.[1] For taste appeal, for economy and "keeping" quality, and for household convenience, meats-in-a-can are advantageous in many respects.

As the comparative data here shown indicate, kitchen-prepared meats and similar meats-in-a-can are closely alike in the amounts of various nutrients they provide.

COMPARATIVE COMPOSITION OF KITCHEN-COOKED AND COMMERCIAL-CANNED MEATS
(Nutrient Amounts per 100 Grams)

	*Kitchen-Cooked Ham[2]	**Canned Ham[3] (Chopped, Cured)	Kitchen-Cooked Beef Round[2]	Canned Roast Beef[2]
Water	50%	50%	59%	60%
Protein	21 Gm.	20 Gm.	27 Gm.	25 Gm.
Fat (ether extract)	28 Gm.	20 Gm.	13 Gm.	13 Gm.
Niacin	4.0 mg.	4.3 mg.	5.5 mg.	4.2 mg.
Riboflavin	0.21 mg.	0.19 mg.	0.22 mg.	0.23 mg.
Thiamine	0.46 mg.	0.40 mg.	0.08 mg.	0.02 mg.

*Values after conversion from 42% to 50% water basis.
**Values after conversion from 58.69% to 50% water basis.

Experimental studies have shown that the processing which meats-in-a-can undergo leads to little if any greater vitamin losses than does home-cooking of similar cuts of meat. In general, meats-in-a-can retain of their original vitamin content approximately:

60 to 80 per cent of thiamine
90 to 100 per cent of riboflavin
90 to 100 per cent of niacin
80 per cent of biotin
70 to 80 per cent of pantothenic acid.[4,5]

During storage for customary periods, at usual warehouse temperatures, meats-in-a-can show little, if any, further vitamin loss except in thiamine. Even thiamine, a highly thermolabile vitamin, was 52 per cent retained in pork-in-a-can after ten months' storage at 80° F. Retention of the vitamin was notably greater when the canned pork was stored at 38° F.

Since meats-in-a-can are thoroughly cooked in processing, they may be consumed as purchased, merely warmed or mildly cooked. When the meat is moderately cooked in preparation for consumption, little or no further loss in vitamins need to occur.

Recent studies show that meats-in-a-can are excellent sources of needed amino acids.[6] The 18 amino acids determined in these studies appeared in similar ratio and amounts in canned beef, pork, and lamb as in the respective fresh or home-cooked meats.

1. Howe, P. E.: Foods of Animal Origin, Handbook of Nutrition, American Medical Association, ed. 2, Philadelphia, The Blakiston Company, 1951, p. 637.

2. Watt, B. K., and Merrill, A. L.: Agricultural Handbook No. 8, United States Department of Agriculture, 1950.

3. Schweigert, B. S.; Bennett, B. A.; Marquette, M.; Scheid, H. E., and McBride, B. H.: Food Res. *17*:56 (Jan.) 1952.

4. Rice, E. E., and Robinson, H. E.: Am. J. Pub. Health *34*:587 (June) 1944.

5. Schweigert, B. S.: Am. Meat Inst. Foundation, Circular No. 8, Nov. 1953.

6. Schweigert, B. S.; Bennett, B. A.; McBride, B. H., and Guthneck, B. T.: J. Am. Dietet. A. *28*:23 (Jan.) 1952.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

American Meat Institute
Main Office, Chicago . . . Members Throughout the United States

from the clinical observations made thus far, it is estimated that **ILOTYCIN** (Erythromycin, Lilly) represents an antibiotic of choice in more than 80 percent of all infections treated by physicians . . .

ILOTYCIN

the original Erythromycin

ELI LILLY AND COMPANY, INDIANAPOLIS 6, INDIANA, U.S.A.

The American Journal of Surgery

A PRACTICAL JOURNAL BUILT ON MERIT

sixty-third year of publication

VOL. 87 JUNE 1954 NUMBER SIX

Editorial

CLINICAL HYPOXIA

IN the summer of 1952 arrangements were made to present a course on the prevention of asphyxial accidents at the University of Colorado Medical School. The speaker arrived in Colorado the afternoon of Saturday, September 13th. As the necessary departmental contacts could not be effected at the time, a tour of the institution was made through the courtesy of the surgical resident. In the course of this inspection the visitor was impressed by the excellence of the University Department of Experimental Hypoxia, which was complete with special laboratory facilities, library and an active polio ward in which respirator patients were receiving specialized treatment. At the completion of the visit the inescapable issue arose: What in the world are you doing here? What have you to add to this splendid assembly of material? The answer to the question shortly emerged.

Assuming that 100 physicians associated with the University were familiar with the findings of this department of experimental hypoxia and that each medical school in the country possessed a similar department with an equal number of interested physicians, the hypothetical 7,900 compared with the national total of 165,000 physicians represents less than 5 per cent. The problem, therefore, the task before us, is to help indoctrinate 95 per cent of the medical profession in the fundamentals of clinical hypoxia.

What about dental schools? It may be assumed that the 5 per cent will not be paralleled by an equal number of dentists. Yet what is being done concerning the hazard of hypoxia in dental offices where the routine use of intravenous pentothal® sodium is practiced?

At the 64th Annual Meeting of the Association of American Medical Colleges held in Atlantic City, October 25–28, 1953, the following statements were released by the Society for the Prevention of Asphyxial Death, Inc.:

Clinical hypoxia is an administrative problem; only the dean has the freedom, the vision, the drive and can direct the "know how" to professional asphyxia consciousness.

Clinical hypoxia is not to be confused with a specific disease or a specific accident. It cuts across the entire field of medicine. The problem is as broad as hemorrhage; as a matter of fact, hemorrhage is a classic form of anemic anoxia.

Clinical hypoxia cannot be departmentalized. To attempt to contain it within the limits of a specialty is to overlook asphyxial accidents in others.

The various facets of clinical hypoxia have in the past been segregated in various departments, i.e., physiology, pharmacology, anesthesiology, obstetrics, pediatrics, internal medicine, etc. The result has left an unrelieved annual mortality exceeding 50,000 a year.

The alternate approach, indoctrination by integration, by what is recognized as "interdepartmental correlation" would seem necessary if the end result of *experimental* hypoxia is to be activated as clinical practice by the intern, the resident and the general practitioner.

The Society for the Prevention of Asphyxial Death, Inc., is presently appealing for a grant of one million dollars, $100,000 a year for ten years, to assist the medical schools to indoctrinate undergraduates, practicing physicians and dentists to prevent and to treat clinical hypoxia.

Experience indicates that intensive indoctrination may be accomplished outside of curriculum hours. More than ninety pilot projects have suggested and have supplied the demand for this information.

Contributions to the grant requested are made payable to the New York Eye and Ear Infirmary, Hypoxia Fund. For almost ten years the New York Eye and Ear Infirmary, through its Post Graduate Medical School established in 1823, has provided the sponsorship and authority which has enabled the Society for the Prevention of Asphyxial Death, Inc., to carry on its vigorous campaign. As a result of this effort many practicing physicians, dentists and more recently medical schools have been alerted to the problem of Clinical Hypoxia. Assistance to continue and to expand this work is thoroughly justified for two reasons: (1) the activity has proven its value over a period of years, (2) the demand for expansion far exceeds the capacity to meet it.

The immediate problem is urgent, namely, to indoctrinate as rapidly as possible 95 per cent of the medical and dental profession, who while not yet asphyxia conscious, are hourly responsible for the prevention and the treatment of impending asphyxial death.

With a view to making instruction easily available to the general practitioner in a short period of time, intensive week-end courses were begun in New York City in 1947. Ninety-one of these courses have now been presented. The course begins on Friday at 1 P.M. and continues until 5:30 P.M. It is resumed and concluded on the following morning. A period of approximately seven hours of intensive instruction is thereby provided. The matriculation fee is fifty dollars.

The material offered includes lectures, slides and a detailed consideration of instruments for the prevention of asphyxia. Anesthetized dogs are brought into the classroom. The class is then taken to the operating room where the airway of an anesthetized patient is demonstrated. In the morning session resuscitation apparatus and the indications for its use are discussed. Human cadavers are provided for practice.

More than 700 students from various parts of the United States have attended these courses. While the sessions in New York City have accepted students from all parts of the country and Canada, many courses have been held in other cities. These have been offered in Columbus, Ohio; Boston; Washington, D. C.; Atlantic City; Baltimore; Denver; San Francisco; Los Angeles and Honolulu. This has been done with the cooperation of the medical schools of Ohio State, Harvard, Georgetown, University of Maryland, University of Colorado, University of California and U. C. L. A., and with the assistance of the Department of Civilian Defense of the State of New Jersey and of the Territory of Hawaii.

Out-of-town courses require a preparatory period of two months and a minimum group of twelve students on the East coast and twenty on the West coast, in order to justify the logistics involved. Courses are given under the auspices of the New York Eye and Ear Infirmary by the Society for the Prevention of Asphyxial Death, Inc. In New York City groups meet the first Friday and Saturday of each month at the New York Academy of Sciences, 2 East 63rd Street.

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Further Experiments on Animals and Man Supporting the Bioelectrical Method of Measuring the Rate of Healing of Wounds

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PREVIOUS publications from this laboratory[3-7] have described the electrical method of measuring the rate of healing of minor abrasions inflicted experimentally on human skin. The purpose of these studies is to determine the possible retarding or accelerating effect of various dressings and drugs on the rate of healing of human wounds. The research reported in this paper had the following objectives: (1) investigation of the great variation which occurs in measurements of wound potentials (these electrical fluctuations were studied by means of a multiple electrode placement method in rats and a statistical analysis of experimental human wounds); (2) comparison of muscle and skin wounds in animals; (3) studies of the reversal of potential which occurs at healing and at death of the animal; (4) measurements of the local effects of external factors such as anesthetic and ions on wound potential; (5) the specific action of chlorophyll on the rate of healing of experimental human wounds; and (6) studies of the electrical potential of a clinical wound.

METHODS

For the animal experiments three measuring instruments were used: a Leeds and Northrup type K potentiometer, a Micromax and a Speedomax recording potentiometer. The accuracy of a measurement could be tested by switching the wound potential on each of the instruments in succession. In some experiments the rat and entire electrode system were placed in a thermostat at 37°C. For human potentials the Micromax recording potentiometer was used. This was taken to the bedside of a patient on a small table on casters and gave satisfactory results. For wounds on the back of rats and for the ulcer on the hospitalized patient, glass tubes filled with saline agar connected the wound to the potentiometer by way of saline cups into which dipped non-polarizable Ag-AgCl electrodes. The potential of the experimental abrasions on the fingertips of healthy humans was measured by dipping the fingertips into cups of saline connected by salt bridges to the Ag-AgCl electrodes leading to the potentiometer. As with humans, the experimental abrasions on the forepaws of rats were made with sterile sandpaper. Rats were anesthetized with ether followed by I.P. 15 mg./kg. pentobarbital as required. The forepaws of the anesthetized rats were placed in two cups of saline and the potential difference between the wounded and intact paw measured as with humans. The potentials of the wound on the back of rats (made by excising skin and, in some cases, muscle) were measured against the intact hindpaw. It was found that rats placed in a jar with ether frequently reopened their wounds during the excitement stage and also died from an overdose of ether. A simple method was devised for administering ether to the wounded rats to soften the excitement stage and to avoid overetherization. A common laboratory glass funnel 20 cm. in diameter was inverted over the animal. The upper narrow tube of the funnel contained cotton to

which ether was added from above. The rat rested on a wire screen placed on a flat board allowing ventilation on the floor of the "cage." At first the rat attempted to escape upward through the narrow tube of the funnel and inhaled ether. The more active the rat, the more anesthetic was inhaled. When the rat became limp and rested on the ventilation screen on the floor, the amount of ether inhaled was less and overdosage was avoided. If the rat recovered, it tried to escape upward again and was reanesthetized. Induction was slow and usually without the excitement stage. Injury to the wound and fatal overdosage were avoided. These points are important in studies of wound potentials in animals who have to be anesthetized daily for long periods so that the rate of healing can be measured. Ether was used to avoid large daily doses of barbiturates which might have some metabolic effect influencing the rate of tissue repair.

A rectangular system of reference points was used for the measurement of wound potential on one forepaw of the rat. The wound potential was measured against the intact forepaw and also against the intact hindpaw on the same side. Also, the potential changes between intact forepaw and intact hindpaw and between the intact hindpaws were measured daily. In this way the wound potential could be corrected for spontaneous changes in the potential of the intact reference point. The rats were weighed at intervals (150 to 200 gm.) and no loss in weight was observed. The diet consisted of Pratt rat pellets and fresh vegetables, fruit juices and U. S. prime beef to eliminate any hypoproteinemia or vitamin deficiency.

RESULTS

The wound potentials made by excising circular areas of skin (1 to 2 cm. in diameter) on the back of the rat were only a few millivolts positive to the intact hindleg. This positivity declined with healing. The wound potential of this type of lesion in animals has been studied by many investigators and we shall not report our data in full except to state that the positivity of skin wounds was confirmed. After measuring the well known positive wound potential in the skin of the rat which averaged about 5 mv., we deepened the wound into the underlying muscle and found an immediate reversal of the wound potential to negativity of 5 to 15 mv. The severity of the wound and the type of tissue involved may be detected by the electrical method. The healing rate of the negative muscle wound potential was much more rapid than that of the positive skin potential. Since our measurements were made at twenty-four-hour intervals, we are unable to report the rate of healing of these superficial muscle wounds in which the negative potential was reversed to positivity by the skin lesion on the next day. In several cases the negative wound potential was found to decrease within an hour and in one animal only 10 per cent of the original negative wound potential was found after twelve hours. Amputation of the rat's leg (through the femur) gave a negative potential of 5 to 20 mv., showing that the negative wound potential dominates the smaller skin potential.

TABLE 1

MAGNITUDE OF INITIAL WOUND POTENTIALS OBTAINED BY EXCORIATING THE PAW OF THE RAT

Rat Number	Potential (mv.)	Room Temperature (°C.)	Electrical Resistance before Wounding (Ohms) R_1	Electrical Resistance after Wounding (Ohms) R_2
12	+14	23.5	100,000	20,000
13	+11	24.5	30,000	3,000
14	+ 9	23.0	90,000	1,000
16	+11	31.0	50,000	5,000
18	+ 6	30.0	70,000	20,000
19	+ 6	31.0	60,000	20,000
20*a* *	+ 8	33.0	50,000	30,000
20*b*	+ 6	37.0	85,000	35,000
22*a*	+ 4	23.0	30,000	20,000
22*b*	+ 5	29.0	13,000	7,000
24	+12	28.0	50,000	40,000

* No. 20*a* gives the data on left forepaw wound of rat 20. No. 20*b* is the right forepaw wound on the same rat 60 days after the first wound. The 20*b* measurement was made in thermostat at 37°C. No. 22*a* and 22*b* are the right forepaw wounds of rat 22 made 50 days apart.

Of the twenty-five animals investigated, half were subjected to dorsal skin wounds and half to sandpaper abrasions on one forepaw. No previous study of skin abrasions on the rat's paw have been published so we wish to report quantitative data. Table 1 displays the wound potentials which averaged 8.3 mv. positive as measured against the opposite uninjured forepaw.

The duration of the paw potentials is shown in Table II. As will be seen in Figures 1 to 5, these potentials form plateaus. In the table the initial potential is divided by the time in days when the curve is descended to the baseline. The end point is taken when the positive wound potential reaches the baseline and becomes negative to the opposite intact forepaw. The magnitude of the initial wounds on the paw ranged from 4 to 17 mv. positive. Healing, as determined by the electrical method, took four to fourteen days. No permanent scars were left on the paws and recovery was complete in all cases. On the first day or two the rat held up the injured paw. Wounds and isolation cages were sterilized with zephiran® daily. After the wound potential had disappeared, the paw appeared normal except for faint pink patches on the fingers, palm and wrist. Other signs of recovery which correlated with the loss of wound potential were the use of the paw in washing the face, feeding and walking. Within thirty minutes after wounding with sandpaper the injured paw swelled to twice the normal size. The paw returned to normal size when the wound potential disappeared.

In experimental abrasions on human fingers we have noted an occasional negative "overshoot" potential which occurs when the wound is healed. In other words, the positive wound potential may become negative by a few millivolts. This reversal of wound potential at the time of healing was also observed in rats. In all eleven animals seven demonstrated reversal, two did not show it and two died in anesthesia before healing was complete. Table III indicates that the negative reversal occurred on the seventh to the fourteenth day (depending on the particular animal) and that the average time of appearance was 10.5 days. The magnitude of the potential averaged 5.7 mv. negative (range 3 to 9 mv.).

The method of recording potentials between the wounded and intact forepaw, between the wounded forepaw and intact ipsilateral hindpaw and between the two intact hindpaws gives a more complete picture than the usual

TABLE II

PERSISTENCE OF POSITIVE WOUND POTENTIAL ON RAT PAWS IN DAYS

Rat Number	Potential (mv.) / Days	Average Loss of Potential per Day (mv.)
12	17/14	1.21
13	11/7	1.58
14	9/6	1.50
18	6/6	1.00
19	6/4	1.50
20*a**	8/8	1.00
20*b*	6/8	0.75
22*a*	4/14	0.28
22*b*	5/12	0.20

* No. 20*a* is the wound on the left forepaw of rat 20; 20*b* is the right forepaw wound of the same rat 60 days after the first wound. No. 22*a* and 22*b* are the right forepaw wounds of rat 22 made 50 days apart. Measurements on rat 20*b* were made in thermostat at 37 ± 1°C.

TABLE III

REVERSAL OF POSITIVE WOUND POTENTIAL ON RAT PAWS AT TIME OF HEALING

Rat Number	Negative Reversal Potential (mv.)	Time since Wounding (Days)	Room Temperature (°C.)	Appearance of Paw
12	−6	14	33.0	Almost normal; faint scar on 1 digit and on wrist
13	−9	9	23.5	Normal; digits slightly pinker than control
14	−7	8	31.0	Digits slightly swollen; small scabs on 2 digits and wrist; rat uses paw to clean face
18	−7	7	32.0	Small scab on wrist and 1 digit
19	−6	13	30.0	Slightly pinker than control
20*b**	−2	10	37.0	Slightly pinker than control
22*a*	−3	13	29.5	Slightly pinker than control

* Measurements on rat 20*b* were made in thermostat at 37 ± 1°C.

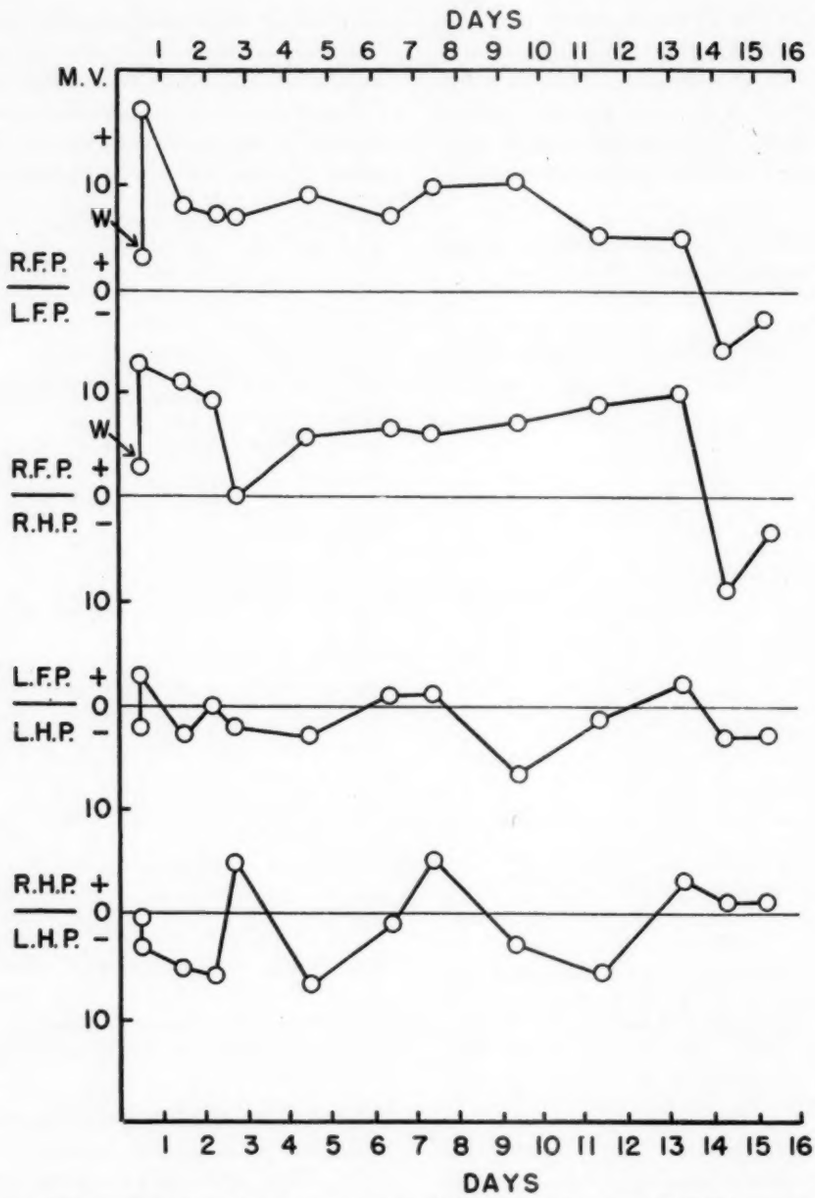

FIG. 1. Wound potentials on paw of rat (paw-to-paw potentials). Right forepaw (R.F.P.) was wounded with sterile sandpaper, indicated by W and arrow. Rat 12. The procedure with this animal will be described in detail to serve as a model for the caption of the next four figures. Each point plotted on the graph represents the average of five to eight readings (obtained during a period of 10 to 30 minutes). Ordinates show the potential of pairs of the four paws of the animal as measured against various reference points or "indifferent" electrodes in this quadrangular method of bioelectrical technic. Paws were withdrawn from the saline cups and the zero tested between each reading. Ordinates: potential in mv. Positive above base line and negative below base line. R.F.P. signifies right forepaw; L.F.P., left forepaw. R.H.P. signifies right hindpaw and L.H.P. left hindpaw. Thus the first reading on the top graph signifies that R.F.P. is 3 mv.+ to L.F.P. The symbol $\frac{\text{R.F.P.}}{\text{L.F.P.}}$ means that the graph shows the sign of the potential R.F.P. as measured against the reference electrode L.F.P. Abscissas in days. In the upper two graphs the symbol W indicates that the right forepaw was wounded with sterile sandpaper to the point of bleeding (under ether and pentobarbital anesthesia). Tips of the fingers received the most damage. As described in the text, the "mirror image" method of correcting variations in wound potential explains the anomalous "dip" in wound potential recorded on the day between R.F.P. and R.H.P. (second graph from the top of the figure). Room temperatures at the time of successive readings were: 1st day 23.5°C., 2nd day 23.5°C., 3rd day 24.5°C., 5th day 21.6°C., 7th day 25.6°C., 8th day 28.5°C., 10th day 23.6°C., 12th day 23.0°C., 14th day 27°C., 15th day 27°C., 16th day 25°C. On the 14th day there were red marks on the wounded forepaw. On the 15th day the paw appeared almost healed and was completely healed on the 16th day.

method of measuring the wound potential against only one point of reference on intact tissue. It was found that many of the fluctuations in the wound potential were actually spontaneous changes in potential of the normal tissue under the "indifferent" electrode. This point is illustrated in Figure 1 (rat 12). The wounded right forepaw was positive against two contacts with normal tissue, the left forepaw and the ipsilateral hindpaw. In both electrode positions the positive wound potential become negative on the fifteenth day when visual inspection showed that the wounded forepaw appeared normal except for a slight pink color. It will be seen in Figure 1 (rat 12) that the positive wound potential measured against the ipsilateral intact hindpaw appears to indicate healing on the third day but no sign of healing is seen in the record of the wound potential from the other point of reference. The explanation of this discrepancy will be found by inspection of the graph showing the spontaneous fluctuations in potential between the ipsilateral hindpaw and the left hindpaw. On the third day when the wound potential dropped to zero the reference point on the hindpaw gave a spontaneous swing to the positive side thus cancelling the wound potential. There are, of course, many unexplained fluctuations in potential on reference points which do not seem to affect the wound potential such as the positive peak of the right hindpaw seen on the eighth day. In Figure 1 (rat 12) it will be seen that the negative reversal associated with healing is technically confirmed by simultaneous negativity of the wound against both reference points which did not show concomitant positive swings.

Figure 1 has illustrated how wound potential may be decreased by a spontaneous fluctuation in potential at the reference electrode. The opposite effect is seen in Figure 2 where a sudden increase in wound potential forms an unexplained positive peak on the third day. Inspection of the graphs will show that the reference point on the opposite forepaw developed a negative peak potential. This type of "mirror image" potential affords the following method of correcting the recorded positive wound potential. (Fig. 2, rat 19.) On the third day the wound potential of the right forepaw shows a peak of 5 mv. positive above the previous potential. This fluctuation corresponds with a simultaneous "dip" to negativity of 4 mv. of left forepaw measured against the left hindpaw. It appears that we should subtract this 4 mv. from the peak potential on the graph showing positivity of the wounded right forepaw. However, this correction factor (the "mirror image") on the left forepaw to left hindpaw circuit may have been influenced by its own reference point. Fortunately we have the potential measured from the intact left hindpaw to intact right hindpaw. In all cases we take the potential change from the potential on the preceding day. Inspection of the graph in Figure 2 will show that the left hindpaw went 1 mv. positive to the right hindpaw, so the correction factor becomes 4 minus 1 equals 3. This corrected factor is shown in Figure 2.

Figure 3 (rat 18) demonstrates a good correspondence between the wound potential measured from two reference points and shows the effect of an accidental wound on the opposite forepaw and also the effect of death. Figures 4 and 5 present data obtained with temperature controlled by an air thermostat at 37 ± 1°C. The rat with the saline cups, salt bridges and electrodes was enclosed in the constant temperature oven when the wound potentials were measured by the Speedomax recording potentiometer. At constant temperature readings the magnitude of the potentials and the great variation were approximately the same as in previous experiments. Each point on the graph is an average of several readings and it was noted that the variation in the individual readings before averaging was less at constant temperature. In Figure 4 an accidental wound shows the usual delay in the healing curve. At constant temperature unexplained fluctuations in potential still remain which cannot always be resolved by the "mirror image" method. A typical unexplained peak is seen in Figure 4, fifteenth day, left hindpaw to right hindpaw. Figure 5 (rat 20) at constant temperature shows good "mirror image" effects. The most urgent problem in all studies of wound potential is to explain the great variation in measurements. This will be illustrated in Figure 5, seventh day, when the wounded right forepaw went 9 mv. in the positive direction after attaining negativity. A genuine increase in positivity is suggested by the 3 mv. positive peak in the wound as measured by the other reference point (right forepaw to left forepaw). At the same time a "mirror image" was presented by the spontaneous negative peak of 7 mv. in the right hindpaw reference point. The correction is therefore 7 mv. and the

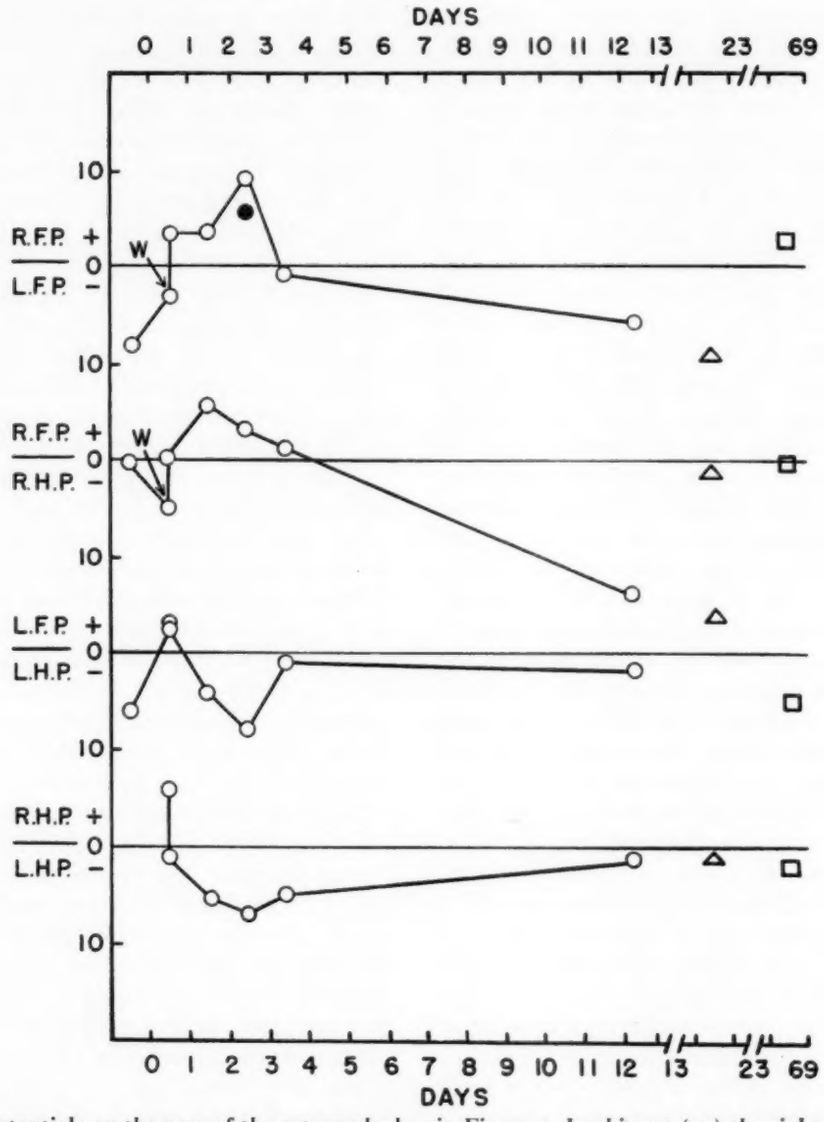

FIG. 2. Wound potentials on the paw of the rat graphed as in Figure 1. In this rat (19) the right forepaw (R.F.P.) was excoriated with sterile sandpaper. Ordinates and abscissas as in Figure 1. Note that the potential was measured the day before wounding. At the end of the graph isolated readings on the 23rd day (triangles) and on the 69th day (squares) are recorded. As explained in the text, the positive peak in the potential of right forepaw to left forepaw on the 3rd day is partly explained by "mirror image" produced by a spontaneous dip to negativity of the reference point (left forepaw). The corrected potential is indicated by the black dot. On the 2nd day the wounded paw was one-third larger than the control and showed red marks on wrist and fingers. On the 3rd and 4th days the paw was slightly swollen with red marks still present. On the 13th day the wounded paw was slightly pinker than the control. On the 23rd day two digits showed faint red patches. On the 69th day both forepaws were identical. Room temperature was recorded at each reading and ranged from 24.0° to 31.0°C.

genuine rise in positivity 3 mv., which yields a value of 10 mv. very close to the observed fluctuation of 9 mv. which might have been mistaken for an exacerbation of the wound potential on the seventh day greater than the potential of the fresh wound.

The positivity of the skin wound in the rat is in some way dependent on the living state of the animal. Wounds on the paws made four to twenty-four hours after death were 2 to 4 mv. negative. (Table IV.) In addition to the negative paw abrasions listed in Table IV, two other animals had circular areas of skin 1.5 cm. in diameter removed from the back after death. Rat 3 gave a wound potential of 6 mv. negative four hours after death (30°C.) and rat 9 had a wound potential of 2 mv. negative three days after death (21.5°C.). In all cases the dead rat

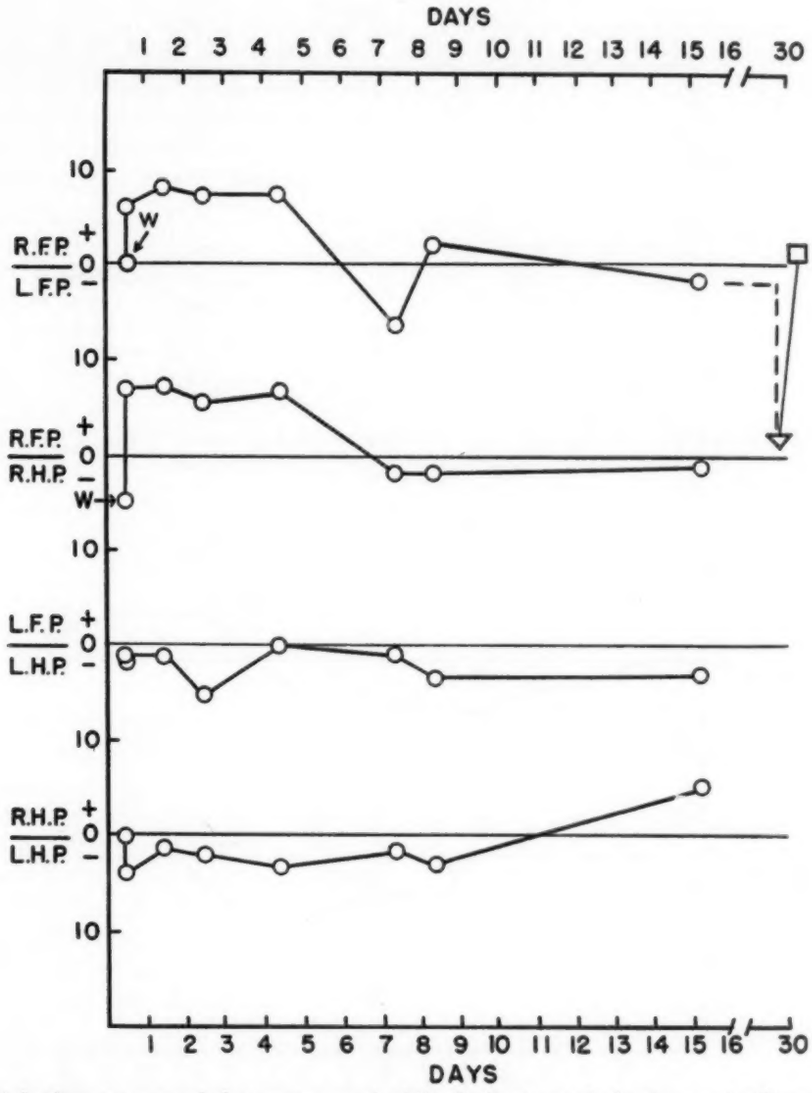

FIG. 3. Rat 18, right forepaw wounded at arrow marked W. Ordinates and abscissas as in Figures 1 and 2. On the 30th day the control left forepaw was accidentally wounded, which caused the potential of the right forepaw (in the top graph) to swing negative (indicated by triangle). This rat died in anesthesia. The wound potential was only 1 mv. negative when measured 3½ hours after death (indicated by square on graph). The above graphs depict the expected potentials in the rat associated with skin wound. The day-to-day potentials between two pairs of unwounded paws fluctuate near zero while the wound potential measured against one of each of these pairs of controls is shifted to positive side until healing occurs. There was considerable hemorrhage from the fresh wound and immediate swelling. On the 2d day the wounded hand was twice its normal size, with many reddish marks. On the 5th day red patches were present and on the 8th day (disappearance of wound potential) the paw appeared normal except for faint scars on wrist and one digit. Room temperature was recorded with each reading (25°–34°C.).

was kept in the refrigerator at 5°C. and allowed to rewarm to room temperature before the wound was made. In some cases a wound was made on the same paw during life and death (after the wound had healed) showing the potential reversal after death. (Table v.) The negative wound potentials of muscle did not reverse to positivity after death but declined in magnitude. The positivity of freshly made skin wounds could be produced for at least half an hour after death (cessation of respiration and heartbeat). In rat 3, removing a circular area of back skin (1.5 cm. in diameter) produced 3 mv. positive twenty-five minutes after death (room temperature 24°C., body temperature 30°C.). The resistance fell from 50,000 to 20,000 ohms. A muscle wound in the same area produced 3 mv. negativity and a

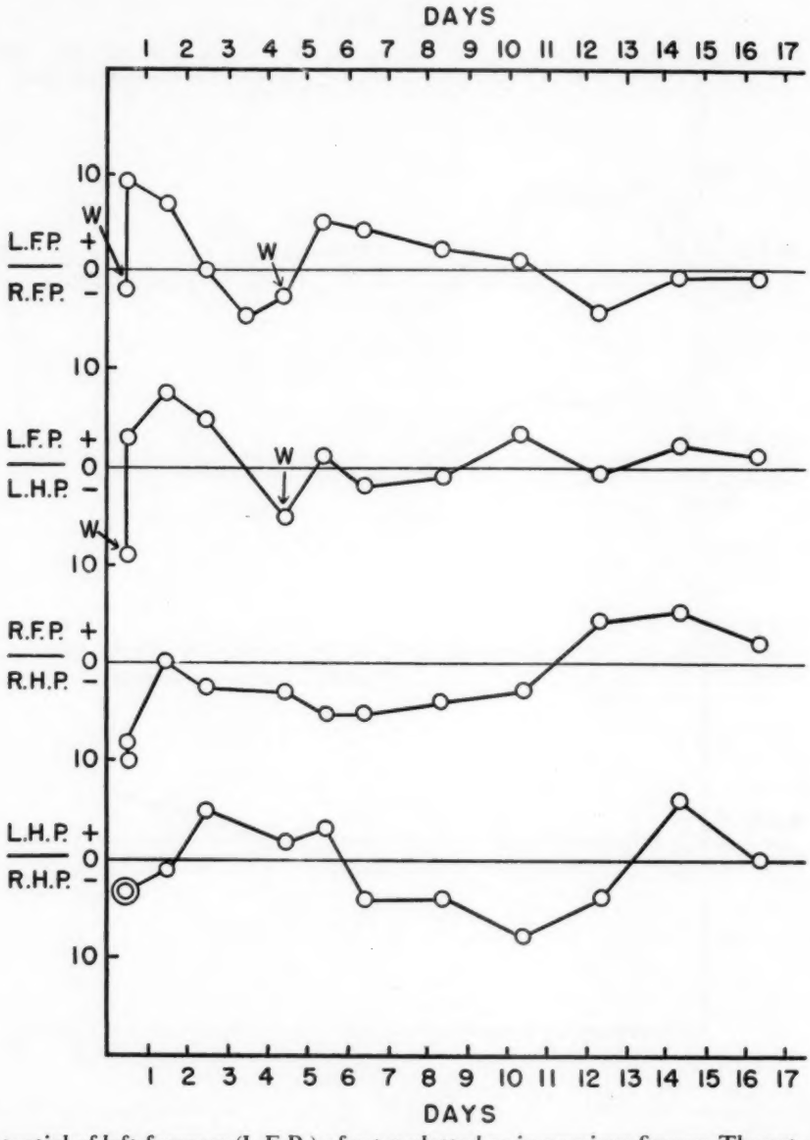

FIG. 4. Wound potential of left forepaw (L.F.P.) of rat 2 plotted as in previous figures. The rat, electrodes and salt cups were placed in thermostatically controlled oven at 37 ± 1°C. at the time of measurement of potential. It will be noted that the spontaneous variations are as great as with uncontrolled temperature. (Figs. 1 to 3.) On the 5th day the left forepaw was accidentally wounded again showing a return to positivity and overcoming the negative overshoot. On the 1st day 50 minutes after wounding the affected paw had swollen to twice the normal size. By the 5th day the paw was slightly swollen and showed a small black and a small red scar. On the 5th day accidental rewounding caused hemorrhage. By the 13th day a small black scar remained on the digit. At this time the wounded paw was used in climbing and in cleaning face which correlated with the decline in wound potential.

further fall in resistance to 10,000 ohms. Four hours after death (body temperature 24°C.) a new skin wound produced 6 mv. negativity showing the reversal after death. A muscle wound made at this time in this area increased the negativity to 7 mv. Before death in this animal a muscle wound gave 19 mv. negative (room temperature 23°C., seventeen days before death).

It has long been known that wound potentials are not as susceptible to alteration by changes in the immediate environment (temperature, chemicals, etc.) as are action potentials (Barnes[2]). There is a voluminous literature on the effects of ions on potential of skin and muscle. However, we thought it desirable to measure the effects of common ions and anesthetics as recorded by our method. Replacing the saline with isotonic KCl had a very slight effect on the negative wound potential

of the rat's leg after amputation. In rat 30 the wound was 15 mv. negative in saline and 16 mv. negative in isotonic KCl. Complete descriptions of these ionic studies will be published elsewhere. In connection with the electrical measurement of wound healing described in this paper we wish to emphasize the fact that wound potential is not markedly altered by changes other than tissue growth or death. A similar invulnerability to ions of human skin abrasions will be described later in this communication. To test the hypothesis that wound potential is not immediately altered by the local application of drugs or chemicals, 1 per cent dibucaine (nupercaine®) was applied to intact skin of rats, regions of excised skin on back and excoriated paws. An insignificant shift to negativity was found in fifty-one experiments involving intact and wounded tissue. The average potential change was 1.25 mv. negative, standard error 0.30 and standard deviation 2.14. This slight shift to negativity was the same on intact and wounded skin. Thus in rat 2 dibucaine produced 1 mv. negativity on the freshly wounded paw and also on the healed paw seven days later. Petrolatum and lanolin had no significant effect. Dibucaine ointment, petrolatum and lanolin increased electrical resistance.

TABLE IV

POTENTIALS OF EXPERIMENTAL WOUNDS MADE ON THE PAWS OF RATS AFTER DEATH

(See also Table v, and Figure 1 for explanation of letters in second column)

Rat Number	Paw Wounded	Wound Potential (mv.)	Hours after Death	Room Temperature (°C.)	Electrical Resistance before Wounding (Ohms) R_1	Electrical Resistance after Wounding (Ohms) R_2
4	L.H.P.	−3	3.5	31.6	40,000	20,000
7	L.F.P.	−2	32.0	29.0	60,000	33,000
12	R.F.P.	−4	5.5	31.5	40,000	15,000
13	L.F.P.	−4	3.5	29.0	60,000	20,000
22	L.F.P.	−3	8.5	29.5	30,000	20,000
23	R.F.P.	−4	24.0	26.0	50,000	30,000
24	R.F.P.	−4	24.0	26.0	50,000	40,000

The remaining part of this paper will describe experiments on wound potentials in human subjects. In a preceding paper[4] we described the decline in negative wound potential of an experimental circular wound 3 cm. in diameter on the human forearm. We wish to describe the wound potential of a leg ulcer in a hospital patient. Figure 6 shows the decline of the positive potential of the ulcer with healing. The potential was 60 mv. positive at the

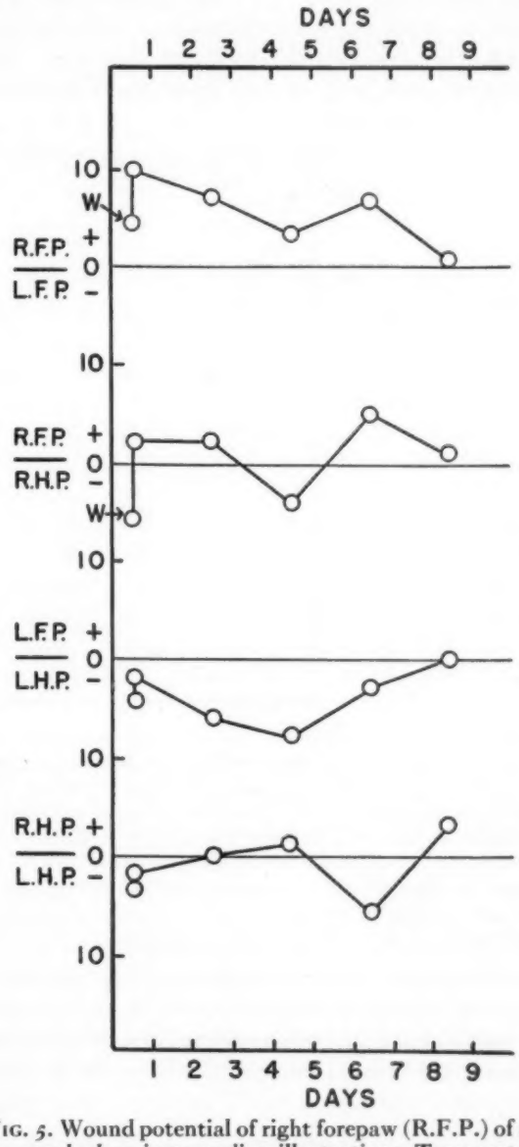

FIG. 5. Wound potential of right forepaw (R.F.P.) of rat 20 graphed as in preceding illustrations. Temperature controlled at 37 ± 1°C. as in the experiment reported in Figure 4. The dip below the base line in R.F.P. to R.H.P. (2nd graph from top) on the 5th day is partially explained by the "mirror image." See text for an analysis of the positive peak of 9 mv. on the wounded right forepaw (R.F.P.) measured against intact right hindpaw (R.H.P.) on the 7th day (2nd graph from top). On the 1st day the wounded paw swelled to twice normal size in 25 minutes after wounding. On the 3rd day the injured paw was somewhat swollen, pink in color and showed red scars on the digits. On one digit the claw was missing. On the 4th day the injured paw was occasionally used. On the 5th day the paw was almost of normal size except for slight swelling of digits. On the 7th day the paw was approaching normal appearance. On the 9th day a few faint scars were present on digits.

beginning of the series of measurements, at which time the wound was 75 by 50 mm. on the malleolus. Over a month later when the ulcer showed pronounced healing and the patient was discharged from the hospital, the potential was only 4 mv. positive. The ulcer was irregular in shape and there was discoloration of adjoining skin which made it difficult to make an accurate measurement of size by visual inspection. Four months later the patient returned with the ulcer open again, giving a large wound potential and appearing as it did at the beginning of the series of measurements. A wide variety of dressings and ointments were used in the hospital and by the patient's physician so that we cannot relate any particular potential reading with a specific ointment. We wish to point out the correlation between the healing of the ulcer and the positive wound potential.

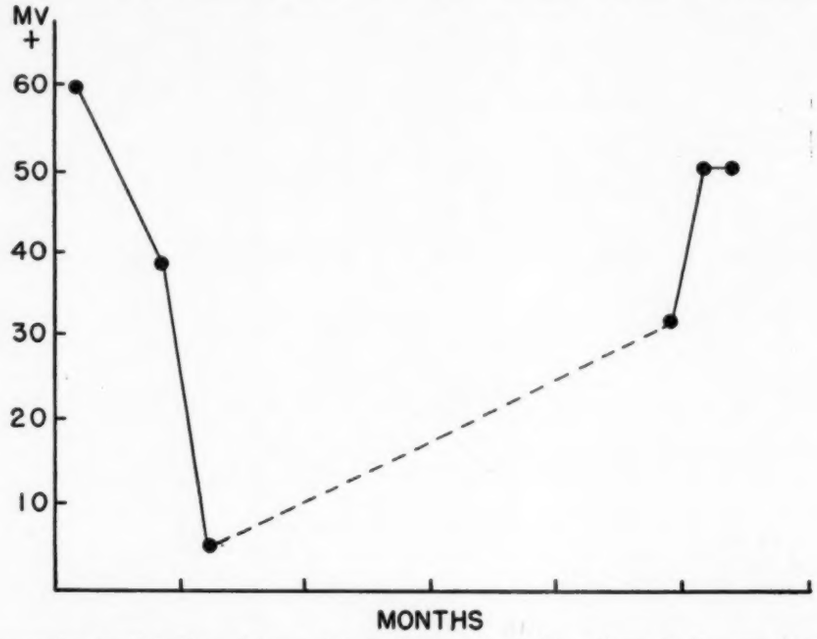

FIG. 6. Positive wound potential of an ulcer on the malleolus of a male patient (P. C., age thirty-five years). The ulcer developed from a ten-year old football injury. Two skin grafts failed. Ordinates: wound potential measured from ipsilateral great toe. Abscissas: time in months. At the beginning of electrical studies the ulcer was oblong in shape 75 by 50 mm. Patient was unable to walk at beginning of this series of measurements. The ulcer healed as indicated by the decline in wound potential and patient was discharged from the hospital at the point of lowest potential. The dotted line represents time when patient was away from hospital. The ulcer became worse again. At the 5th month the patient returned to the clinic, and electrical measurements showed that the positive wound potential had returned and the appearance of the ulcer resembled that at the beginning of the study.

TABLE V

POTENTIALS OF FRESH WOUNDS MADE ON THE SAME PAW OF THE SAME RAT BEFORE AND AFTER DEATH

Rat Number	Potential Alive (mv.)	Room Temperature (°C.)	R_1† (Ohms)	R_2 (Ohms)	Time Interval (Days)	Potential Dead (mv.)	Room Temperature (°C.)	Hours Dead	R_1 (Ohms)	R_2 (Ohms)
12	+17	23.5	.....	.....	43	−4	31.5	5.5	40,000	15,000
13	+ 7	24.5	30,000	5,000	25	−4	29.0	3.5	60,000	20,000
22*	+ 9	25.0	30,000	20,000	..	...	....	....	.....	.....
22	+ 6	29.0	10,000	3,000	12	−3	29.5	8.5	16,000	6,000
24	+12	28.0	50,000	40,000	45	−4	26.0	24.0	22,000	16,000

* Note: Rat 22 was wounded twice when alive in an interval of 3 mo.

† See Table IV for explanation of R_1 and R_2.

In another case we found a potential of 40 mv. positive on the back of the hand two months after a burn by an ether explosion. A skin graft had been made but was considered unsatisfactory by the surgeon. The grafted skin had lost its elasticity and a new graft was planned. Thus the unhealthy state of the grafted skin was correlated with a fairly large positive potential.

The experimental abrasions of human fingers gave wound potentials which were not markedly affected by changes in the chemical environment. Isotonic KCl, $CaCl_2$ and $MgCl_2$ did not produce large changes in potential compared with saline. Also, chloroform and ether did not depress the potentials but on the contrary produced a slight increase, probably by irritation. The following experiment is typical of the effect of chloroform and ether. On a freshly made abrasion on the fingertip the following wound potentials were recorded by successively dipping the wound into saline connected to the potentiometer: +20 mv., +22 mv., +21 mv. and +20 mv. The wound was then placed in chloroform and gave a potential of +23 mv. After washing with saline, the wound potentials were +18 mv., +17 mv. and +18 mv. The wound was then placed in ether and the potential was +21 mv. Three successive readings in NaCl were +19 mv., +20 mv. and +19 mv.

It is fortunate for the electrical method of measuring wound healing that the potential is not easily influenced by changes in the immediate chemical environment. Only regeneration of tissue has a pronounced effect on wound potential. This affords an objective method for testing drugs or dressings used clinically on human wounds. Of numerous substances tested to date, chlorophyll derivatives appear to give the best results when applied to experimental abrasions. We now have a sufficient number of cases to apply statistical analysis for significance of the results. To date, the average rate of healing or per cent wound potential loss per hour is 2.64 per cent for experimental wounds treated with 0.5 per cent Chloresium N.N.R.® compared with 1.58 per cent per hour for controls treated with the ointment vehicle without chlorophyll (on the same hand at the same time). Chloresium is composed of water-soluble derivatives of chlorophyll a. This series included seventy-six wounds in nineteen experiments. The standard error for the average rate with chloresium (2.64 per cent) is calculated to be 0.417 and the standard deviation 1.82. The standard error for the rate on controls (1.58 per cent) is 0.253 and the standard deviation 1.10. The t value is 2.17 and P value is 0.04. This statistical analysis shows that the increased healing rate produced by chloresium is significant.

COMMENTS

The small magnitude of the wound potentials and the wide variation in the electrical potential of wound and intact skin were characteristic of our experimental wounds made by excising circular areas in the skin of rats. Fardon, Judd and Prince[19] reported that potentials from wounds consisting of two crossed incisions 2 mm. in length made on the back skin of mice showed spontaneous changes. Burrows, Iball and Roe[12] suggested that the uncertain results obtained in bioelectrical studies of wounds in the skin of rats may be ascribed to interaction of positive skin wound potentials and negative muscle wound potentials (for example from the underlying panniculus carnosus). By wounding first the skin and then underlying muscle in rats we have shown that this apparent discrepancy between the positive skin wound and the negative muscle wound may be used as an indicator of the depth of the wound and the kind of tissue wounded. Part of the wound potential of rat muscle decreases very rapidly (within the first thirty minutes), resembling an action current.[32] Since the muscle wounds heal within twenty-four hours in the rat while the area of excised skin gives a long-lasting wound potential, the graphs of the sign of the potential of a wound involving skin and underlying muscle show a reversal from negative to positive on the second day. Abrasions made with sterile sandpaper on the paws of the rat were found to give less variable wound potential curves than wounds made by excising areas of skin on the back. The method of multiple electrode placement illustrated in Figures 1 to 5 explains some of the spontaneous changes in wound potentials on the paws of rats. The paw wound which involves chiefly the fingers is relatively free from muscle potential and moreover is analogous to the fingertip abrasions on the human subjects. The fall in electrical resistance on wounding (Table 1) may be used as a measure

of healing and will be described in a future publication. Abrasions on the human fingers give wound potentials five times the magnitude of similar wounds on the rat's paw and are more suitable for testing the effects of healing agents. Moreover, animal skin is histologically and physiologically quite different from human skin. Healing agents for clinical use should be tested on experimental human skin wounds.

The electrical method of measuring the rate of healing of wounds has many advantages over the visual inspection method used many years ago by Carrel and Hartman[14] and Du Noüy,[17,18] and revived from time to time by Smelo,[28] Moss et al.[25] and Homburger.[22] Carrel and Hartmann[14] state that errors of visual measurement of wounds in animals are produced by changes in position of the animal. In the visual method applied to human wounds the same authors admit that errors arise from lack of distinctness in the outer edge of the cicatrix. They state that it was necessary to select wounds in which the measurements could be made with accuracy. The recent report by Moss et al.[25] on the old Du Noüy method requires further comment. It is doubtful if a freehand sketch of the wound can furnish a scientific measurement of wound healing. Moreover, it is difficult to understand the significance of the term "retarded healing" in many cases reported in Table 1 of Moss et al. For example, a forty-four year old ulcer decreased in area from 3.94 to 1.80 sq. cm. during twenty-five days of chloresium treatment, which was recorded as "retarded healing." This conclusion is based on the expected rate of healing calculated on the Du Noüy formula. Instead of direct measurement of a control lesion of the same age and size, Moss et al.[25] compare the clinical healing with the imaginary "ghost" healing calculated on paper from an oversimplified formula. Du Noüy[17] states, "the date of healing could be predicted 90 days in advance" but he gives no statistical analysis to support this claim. Du Noüy[17,18] developed two formulas. In the first method the rate of healing of a wound is investigated by taking two measurements three or four days apart in order to calculate the area it should have four, ten, twenty or thirty days later. Du Noüy's second method gives a "purely theoretical" estimation of how fast a wound should heal and it "then becomes unnecessary to take two measurements of the wound 4 days apart" to obtain the constant characteristic of this particular wound. It is clear that Moss et al.[25] used the wrong Du Noüy formula, which is the second "purely theoretical one." Obviously, to test the effect of a drug on a particular wound the constant for this wound before treatment should be obtained (Du Noüy's first formula). Needham[26] has recently criticized the Du Noüy formula as an oversimplification. Moss et al.[25] point out that many important variables are neglected in the Du Noüy formula such as general status of the patient; hypoproteinemia, vitamin deficiency (A, C, or K), electrolyte and water balance, anemia or systemic infection, tissue trauma, presence of devitalized tissue at the wound site, local temperature and integrity of the blood supply. Recently other factors influencing wound healing in animals have been reported.

Findlay[20] finds that vitamin B_{12} promotes healing. According to Williamson and Fromm[35] the rate of healing is a function of the cystine content of the wound. Pirani et al.[27] have described the effect of DCA on granulation. Chassin et al.[15] find that stress delays healing in rats (also Marshall[23]). All of these factors are automatically cancelled out in the bioelectrical method of measuring the rate of healing of experimental wounds since treated and untreated control wounds are made on the same hand of the same person at the same time. The curve of the Du Noüy equation, however, represents the latter part (major phase of declining growth rate) of a sigmoid curve of growth which was known to Spalanzani in 1768.[31] Auerbach[1] suggests that the last or slowing phase of the healing curve may be produced by liberation of an inhibiting factor. A second fresh wound made on the same animal is inhibited during the declining phase of a previous wound. The bioelectrical method also yields a curve of this form.[4] (Fig. 4.) The great variation in the rate of healing in individual wounds does not permit testing healing drugs by comparison of a single treated wound with this theoretic curve. Controls must be run as in the bioelectrical method. The Du Noüy equation is corrected for only two variables, namely, the age of the patient (not for the age of the wound) and for the size of the wound. In the bioelectrical method applied to experimental abrasions on human skin we attempt to adjust the abrasions to the same degree of wounding at the start by excoriating each wound until

the controls and the wounds to be treated give the same wound potential in millivolts.

The method of measuring wound healing by determination of the tensile strength of wounds by tearing open the wound[34] may be used on the back skin wounds in rats where wound potentials are of low magnitude; but this method suffers from the defect that a series of measurements cannot be made on the same animal. However, this method has confirmed the clinical impression that a secondary wound gains in strength more rapidly than a primary wound.

The greater magnitude of the potential of human wounds compared with those of animals is illustrated by our study of a leg ulcer on a patient for six months. This wound produced its own control by healing and then reverting to the original pathologic condition. Melchoir et al.[24] reported positive electrical potential in human clinical granulating wounds which they believed was an "action current." Beck[8] observed that the positive wound potential decreased when the electrode was moved from a granulating area to a zone where epithelization had begun. Beck studied the electrical wound potential of both dogs and human patients. Burr et al.[10,11] measured the healing rate by the bioelectrical method in twenty-five instances of operative procedures in man and also in experimental wounds in guinea pigs. Crane[16] confirmed our bioelectrical method of measuring wound potential in an investigation of a human clinical wound which required sutures.

Finally we wish to comment on our results with "chlorophyll." As pointed out by the botanist, Thimann,[33] authors should be more specific about the term "chlorophyll." The results reported in this paper were obtained with chloresium N.N.R., water-soluble derivatives of chlorophyll which consist chiefly of the copper complex of the sodium and potassium salts of saponified chlorophyll. Botanists and chemists unfamiliar with the scientific literture on wound healing have published statements which question the effects of chlorophyll derivatives on the rate of wound healing. It is hoped that the bibliography appended to this paper may help to clarify the confusion. Bürgi[9] experimented on rabbits and guinea pigs. Wounds 2 cm. in diameter were made by removing circular areas of skin. Application of chlorophyll derivatives accelerated healing as compared with untreated controls (judged by photographs). Gruskin,[21] a pioneer in the use of chlorophyll derivatives in this country, reported favorable results in a series of 1,200 clinical cases. Smith and Livingston[29,30] reported that chlorophyll promoted healing in a large series of experimental wounds on animals, but we deplore their use of the term "dramatic." Carleson and Garsten[13] reported the accelerating effect of septofyllin, a chlorophyll derivative, in a series of 130 human clinical wounds. The results on experimental human wounds treated with chloresium reported in this paper offered statistically verified evidence that derivatives of chlorophyll accelerate the healing of wounds.

SUMMARY

1. A comparative study of the wounds of white rats and man shows the greater magnitude of wound potential in man. For this reason, healing agents should be tested on human experimental wounds when the bioelectrical method is used.

2. In rats the kind of tissue involved in an experimental wound can be detected by the electrical method, positivity for wounded skin and negativity for wounded muscle.

3. Wound potentials are not susceptible to alterations in the environment such as the application of general or local anesthetics or ions. On the other hand, the process of healing is related to the magnitude of the wound potential.

4. Some of the spontaneous fluctuations in wound potential appear to arise from an internal source. Daily measurements of wound potentials made in a thermostat at 37°C. continue to show variations in magnitude. A method using four electrode placements in rats showed that variations of potential at the reference electrode may raise or lower the apparent wound potential.

5. Skin wounds were made by excoriation on the forepaws of rats to compare with the experimental wound potentials made on human fingers as described in previous reports. As in man, the positivity of the wound potential disappeared at the time of healing. The rat wounds showed a reversal to negativity which is less frequently found in man.

6. In rats the positive wound potential is not found on wounding the skin after death.

In dead rats a small negative potential is produced by wounding the skin.

7. The wound potential of an ulcer on a hospitalized patient decreased with healing.

8. Drugs used in the treatment of wounds can be tested for possible accelerating effect on the rate of healing by measurements of the rate of decline of positive wound potential of experimental abrasions on human fingertips. For example, wounds treated with 0.5 per cent chloresium ointment N.N.R. showed a healing index or per cent wound potential lost per hour of 2.64 compared with 1.82 for controls. The accelerating effect of chloresium is statistically significant.

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The Importance of Anatomic Precision in Differential Sacral Neurotomy for "Cord Bladder"

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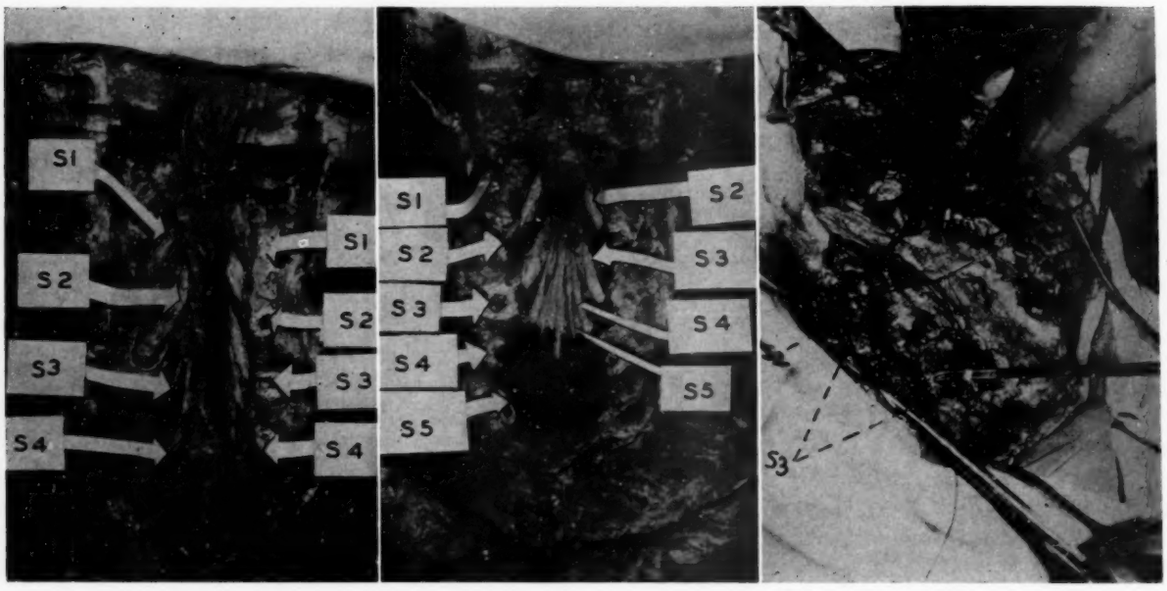
WHEN spastic bladder disturbances of spinal cord origin are present in patients who are ambulatory, some surgical method other than bilateral anterior intradural root section (T12-S5) must be utilized if any operation is to be done at all; otherwise, the patient will be paraplegic postoperatively.

We are indebted to Meirowsky and his associates[1,2] for demonstrating conclusively that differential sacral neurotomy is (via transsacral operation) as effective in establishing bladder automaticity as is anterior root section intradurally in paraplegic patients. As stressed by Bors[3] still other methods utilized for bladder dysfunction of cord origin are pudendal neurectomy (the pudendal nerve derives its fibres from the ventral branches of the second, third and fourth sacral nerves) and transurethral prostatic resection. Neurogenic bladders resulting from cord lesions above the conus differ functionally from those with lesions at or below the conus. The purpose of this paper is not to discuss the indications for these different surgical procedures, as they have been covered adequately by previous writers,[1–3] but, rather, to stress the importance of certain anatomic characteristics of the sacral nerves as exposed through the sacral foramina in differential sacral neurotomy.

Although Meirowsky and his associates[2] in their paper on differential sacral neurotomy as an operative method for the relief of urinary retention with bladder spasticity were utilizing it for an entirely paraplegic post-traumatic patient (their patient later improved after decompressive laminectomy), we had an opportunity recently in our clinic to apply the procedure to an ambulatory patient. This patient had had an unusual virus infection of the entire central nervous system in 1947 which we believed was myeloencephalitis. At that time he could not move any extremity for several weeks. It was an extremely severe illness and bilateral choked disks also developed as well as a sensory level to pin-prick over the trunk anteriorally at the 7th thoracic dermatome bilaterally, the patient then being thirty-five years old. At that time a partial subarachnoid block was present as shown by spinal puncture; the fluid was xanthochromic and showed 49 white cells per high-power field, most of which were lymphocytes, and 120 mg. per cent of protein. The diagnosis was severe encephalomyelitis involving the brain and cord. This resulted eventually in an extremely spastic state of the lower extremities and bladder suggesting, very closely, multiple sclerosis but he was still ambulatory when seen by us in January, 1952, for neurologic analysis of the urinary difficulty and suggestions for its surgical correction.

The patient's chief difficulty then was bladder dysfunction, particularly the emptying of his bladder satisfactorily. He had to be catheterized every morning. As reported by a urologist (April, 1952), the patient had a competent automatic bladder but could not empty it as there was increased tone of the external voluntary sphincter. During certain periods of relaxation, notably bowel movements, he was able to empty his bladder spontaneously. Usually, there was incontinence with overflow, dribbling and poor emptying. It was discovered that after novocain injection of sacral roots 2 and 4 bilaterally through the sacral foramina, when his bladder was full, he could temporarily void a satisfactory stream and empty his bladder with a small residual urine. This was proven on three different occasions but failed to occur when sacral roots 2 and 4 were injected bilaterally with saline instead of procaine, as a control test. It was the



1 2 3

FIG. 1. Cadaver specimen in the first stages of dissection showing the sacral roots S1, 2, 3 and 4 bilaterally at the enlarged foramina. It can be seen on the left side, for instance, that S3 root is very close to S2 root at the second sacral foramen.

FIG. 2. Cadaver specimen at later stage of dissection. The entire sacral group of nerve roots is exposed, the sacral canal having been unroofed entirely. The foramina are no longer in evidence as clearly as in Figure 1. It can be seen, on the right side, that sacral roots 3 and 4 are quite close together at a certain position of their course and one could easily lift up one nerve thinking it was the other when a nerve hook was introduced deeply through an enlarged sacral foramen.

FIG. 3. Photograph of S2 and 3 nerve roots exposed at operation (left side). Note that at the upper foramen (S2), S2 and S3 nerve roots are extremely close together, S3 being, of course, medial to S2 at the second sacral foramen. It would be extremely easy with a nerve hook casually introduced deeply and blindly through this foramen to pick up S3 when S2 might be the one that one wanted to divide.

urologist's opinion that appropriate bilateral sacral nerve divisions should be carried out, as indicated by the novocain blocks.

As the patient was ambulatory, we did not wish to carry out the first recommended procedure of Meirowsky and his associates,[1] namely, extensive intradural anterior rhizotomy from T12 to S5 roots bilaterally, as he would certainly be paraplegic thereafter. Accordingly, the second procedure,[2] that of sacral neurotomy of S2 and 4 roots bilaterally, was determined upon after novocain injection of these roots had shown satisfactory emptying of the bladder with residual urine of not more than 50 cc. The operation was satisfactorily carried out on July 2, 1952, under endotracheal nitrous-oxide-ether anesthesia, but during the course of the operation, and in subsequent studies in the anatomic laboratory, certain interesting anatomic facts were demonstrated which form the subject of and the reasons for this paper.

The nerve roots sectioned in our patient were the 2nd and 4th sacral bilaterally. Postoperatively, he voided satisfactorily in the erect position although there was still some difficulty while lying in bed. It was thought by all observers that there was marked improvement in bladder function in the erect position. Postoperatively no catheterizations were required. Preoperatively he had required daily catheterization and occasionally had had severe genitourinary infection with a septic type of temperature for several days. These bouts of infection constituted one of the main indications for operation on the sacral nerve roots.

It was demonstrated by anatomic dissections in cadavers that the combined anterior and posterior sacral nerve roots 2, 3 and 4, at the appropriate foramina, are easily picked up on a nerve hook when exposed by the technic recommended by Meirowsky and his associates.[2] The point to be emphasized at this

time, however, is that sacral roots 2 and 3, for instance, or sacral roots 3 and 4, are extremely close together at any one foramen. (Figs. 1 to 4.) This anatomic fact leads one to believe that novocain injection of, let us say, S2 and 4 roots, as one might do in an individual patient, might very well result in the actual injection of S2, 3, 4 and 5 roots because the needle inserted percutaneously would have great difficulty in picking out one nerve only at any individual foramen. Caution is therefore advised in stating that preoperative percutaneous injection of foramina S2 and S4, for example, has blocked with novocain only those particular nerves. Likewise, and even more important, at the time of operation, as one sees in the illustrations (especially Fig. 4), sacral roots 2 and 3 at sacral foramen 2, for instance, are extremely close together, the sacral 3 root at that particular foramen being medial to sacral 2 root.

To the occasional operator, and it is for this reason chiefly that this clinical note is being made, it is important to enlarge the foramen sufficiently with a Kerrison punch or similar instrument so as to be certain that the two adjacent nerve roots are identified at any individual foramen and only the one that one wishes to divide is actually divided between silver clips. It would be quite easy for one not accustomed to the operation to insert a nerve hook into the unenlarged foramen, sometimes as far anterior as 1½ cm. from the bony edge, and rather blindly pick up a nerve root thinking it was S2 whereas it could easily be S3 and so on as one inspects the several sacral foramina. This note is a plea, then, for anatomic accuracy in doing differential sacral neurotomy to establish bladder automaticity in certain cases of cord bladder in which there is considerable residual urine present, and increase in bladder and sphincter tone, and which is temporarily relieved by bilateral differential percutaneous novocain block of the nerves in the sacral foramina. This is an extremely valuable procedure, without doubt, in selected cases of bladder dysfunction and we believe it is important to stress that accuracy in differential section of nerve roots can never be emphasized too much. This is particularly true in nerve root operations at the lower end of the spinal cord, cauda equina and the even more peripheral sacral nerve roots at the sacral foramina.

So far as novocain injection of the individual sacral nerves preoperatively by the percutaneous technic is concerned, if one wishes to inject S2 root at the second sacral foramen, for instance, the needle should be directed quite laterally or one will almost certainly inject S3 root also and receive a false impression as to which nerve has actually been injected. In fact, it seems to me that it would be quite difficult in most instances to be certain that one was injecting percutaneously only one sacral root at any particular foramen. The injection technic and the operation are technically without great difficulty and, with the anatomic points stressed previously, should produce excellent results in selected cases.

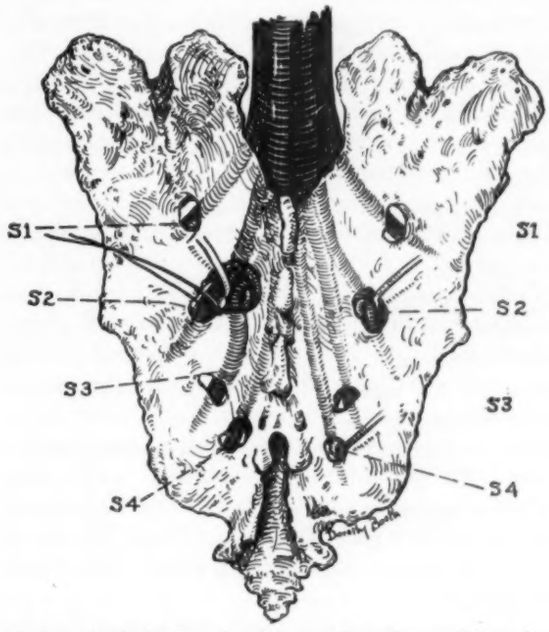


FIG. 4. Artist's drawing illustrating the problem discussed in the paper. At the enlarged foramen at S2 on the left side can be seen the very close approximation of S2 and 3 roots as shown in the photograph of the nerves made at operation. (Fig. 3.) When the nerves lie quite deep, even 1½ cm. beneath or anterior to the foraminal level, it is very easy to pick up the wrong nerve by turning the hook in different directions. Also, when one injects novocain preoperatively, it would be extremely easy and perhaps impossible to inject only one of the two nerves (S2 and 3) shown at the second sacral foramen on the left side; therefore, interpretation of preoperative novocain injections at certain sacral foramina must be done with great caution.

As stressed by Prather[4] cases with partial cord injury, as in our patient with a post-inflammatory lesion of the central nervous system, show some evidence of voluntary motor

power and sensory appreciation below the level of the lesion, although this may not be evident for several months after injury or inflammation of the cord. It is to be hoped that patients with partial cord lesions will progress to a third or final stage of recovery characterized by voluntary micturition sufficient to produce satisfactory emptying of the bladder. The necessity for manual pressure over the lower abdomen during micturition, so marked a symptom in our patient, straining during voiding and diminished sensation from the bladder region may be accompanying features, but there is voluntary control in the third stage of recovery. The organ under these circumstances might be termed a "voluntary neurogenic bladder." Bladder capacity is within normal limits and bladder residual is variable. It is in such cases, whose findings were typically present in our patient, that a differential bilateral sacral neurotomy through the indicated sacral foramina is the operation of choice, particularly when the patient is ambulatory and when bilateral anterior rhizotomy intradurally would, of course, be contraindicated (T12 to S5 inclusive) because of the resultant inevitable paraplegia.

The two methods for improving bladder function after spinal cord injury or disease are anterior rhizotomy of T12-S5 intradurally and multiple sacral neurotomy bilaterally, through the sacral foramina.[5] The latter method is indicated in patients who are not paraplegic, although it can also be used in paraplegics who do not have appreciable flexor spasms of the legs. Such an automatic bladder should be predicted and proved by testing with novocain blocks of the sacral nerves percutaneously before neurosurgical intervention. Relaxation of the peroneal muscles is essential for initiation of micturition. The rationale of either of these procedures is that the interruption of the sacral reflex arc by intradural rhizotomy or by sacral neurotomy eliminates the spasticity of the peroneal muscles. It is essentially a dysfunction of the sacral reflex arc which results in urinary retention in these patients.

In deciding whether either of these two surgical procedures would be helpful, it is important to check the ability of the patient to void without straining both before and after bilateral novocain injection of the sacral roots, and to determine bladder volume and also residual urine before and after novocain injection has been carried out. Preferably, the residual urine should not exceed 50 cc. after novocain block, if operation is to be helpful. If the novocain block of S2, 3 and 4 roots bilaterally, or combinations of these three roots, results in a bladder capacity of 300 to 500 cc. of urine, voiding at will without incontinence, and with a residual urine of not more than 40 to 50 cc., operation (bilateral sacral neurotomy) should be carried out. This procedure is particularly applicable in patients with incomplete cord lesions, as in our patient with a severe virus infection of the cord and brain. Before deciding which sacral nerve roots should be divided in the individual patient, a combination of S2 and 3, of S2 and 4, or of S2, 3 and 4 bilaterally, should be injected percutaneously with novocain and tests made as indicated previously. In one of their cases Meirowsky et al.[5] found, for instance, that by injection of S3 and S4 nerves bilaterally, most relaxation of the bladder occurred; they therefore proceeded with surgical division of S3 and 4 roots bilaterally at those foramina, with excellent results. It is, however, a question whether they could be entirely certain that only S3 and 4 sacral roots were injected percutaneously with novocain through those foramina in the initial tests and not sacral roots 3, 4 and 5, as sacral roots 3 and 4 are very close together at the third sacral foramen and sacral roots 4 and 5 are quite close together at S4 foramen. This is the purpose of the present paper to demonstrate that (1) the percutaneous injection of the sacral nerve roots with novocain and (2) the division of the roots surgically at the sacral foramina must be done with anatomic precision or one might be injecting or dividing the wrong root or roots.

French[6] states that (in speaking of paraplegic cases entirely) pudendal neurectomy has relieved patients with a large amount of residual urine after a transurethral bladder neck resection has failed. Pudendal neurectomy can be performed on patients who respond temporarily to repeated pudendal nerve anesthesia. Other neurectomies, such as presacral neurectomy, resection of the anterior roots of the third sacral nerve or denervation of the bladder by cutting of the anterior and posterior roots of the 2nd, 3rd and 4th sacral nerves intradurally, are of historic and theoretic interest.[3] Meirowsky and associates[1] quote Heimburger, Freeman and Wilde[7] as stating, from clinical investigation, that they concluded that the anterior root

of the third sacral nerve rendered dominant innervation to the human bladder. They further showed that selected cases of neurogenic bladder, characterized chiefly by urinary retention, can be treated successfully by bilateral block of the third sacral roots with novocain solution or with alcohol injected trans-sacrally.

Meirowsky and associates[2] concluded as follows: "With the intent of finding an operative method technically easier and less extensive than the classical intradural rhizotomy within the spinal canal, we have described a method of sacral neurotomy by which any number of anterior and posterior sacral nerves can be sectioned through the sacral foramina. In our indications for this operation we have been guided by preoperative multiple sacral nerve novocain blocks. While in some cases complete elimination of the segmental sacral innervation results in bladder automaticity, there are other cases in which only *differential* sacral neurotomy is required. The necessary extent of the segmental denervation can be determined by differential sacral novocain blocks, simulating, *temporarily*, a status of denervation [such as would be accomplished *permanently* by section of these nerve roots at the sacral foramina]. Results with this method, however, are doubtful whenever the sacral novocain blocks do not result in complete bladder evacuation *at will* [with low residual urine]." The optimum desired effect is the establishment of bladder automaticity by differential sacral neurotomy in ambulatory patients, and in those individuals who have such a bladder this procedure (bilateral multiple trans-sacral neurotomy) is the method of choice. Muellner[8] emphasized that dysfunction of the muscles of the pelvic floor (marked spasticity) has a direct bearing on man's ability to stop and start the urinary stream at will. It is believed that in our patient particularly, who strongly resembled (clinically) multiple sclerosis, this was the essential difficulty with his bladder function and it was relieved by division of S2 and 4 roots bilaterally through the appropriate sacral foramina. Muellner also stressed that a normally functioning levator ani is necessary for normal bladder function. In multiple sclerosis, for example, there is definite dysfunction of the levator ani muscle, in some cases, at least.

SUMMARY

Differential sacral neurotomy is believed to be a very satisfactory procedure for achieving an automatic bladder in spastic lesions of the spinal cord either post-traumatically or after non-traumatic cord lesions such as severe myeloencephalitis. Attention is called to certain anatomic facts with respect to the close approximation of the sacral roots especially at the S2, 3 and 4 foramina in the sacrum as shown by anatomic dissections in the cadaver, and also at operation. The practical application of these facts is that great care must be taken both in interpreting the effects of preoperative novocain injections of the different sacral foramina and nerves, and also in searching for the proper nerve to divide surgically in the individual sacral foramen following novocain injection. The surgeon must take care that, through error, the wrong nerve is not injected or divided. By following certain simple anatomic observations, such errors can be readily avoided.

These observations are directed particularly to the occasional operator in this type of case who might otherwise be led astray in interpreting the result of novocain injections and in identifying the appropriate nerve roots for surgical division at the individual sacral foramina.

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Intraspinal Tumor, a Difficult Diagnosis

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MANY diseases of the nervous system can be confused with spinal cord tumor. Bailey and Craig,[1] writing on intraspinal meningiomas, list multiple sclerosis, arteriosclerosis of the nervous system, subacute-combined sclerosis, peripheral neuritis, and "spinal syphilis." That such confusion does occur and that patients are still brought to definitive treatment late in the course of their disease because of it, is attested by the following representative cases:

CASE REPORTS

Multiple Sclerosis

CASE 1. P. B., a thirty-four year old white man, presented himself for examination in a wheelchair, completely unable to walk. Eighteen months before examination a feeling "like my legs were going to sleep" began in both lower extremities, worse on the left. Starting in the feet, it began to progress up the legs. Next he noticed a stumbling, unsteady gait and, soon thereafter, difficulty in starting the urinary stream. At this juncture he consulted a physician who declared the disease multiple sclerosis. Furthermore, a considerable remission appeared to confirm the opinion. After four months he again became able to walk, ceased staggering and regained urinary control.

Pain now appeared in the low back for the first time, and then only on bending. Soon his condition began gradually to deteriorate and "strange sensations" occurred up to the waist. He was recommended to a diagnostician, who confirmed the first opinion of multiple sclerosis. No spinal tap had so far been done. Some hardness of hearing in the right ear and "weak eyes" of long duration perhaps suggested intracranial as well as spinal involvement.

At neurosurgical examination some one and a half years after onset the patient was completely unable to support himself on his legs, which were spastic and very weak. There was marked hypalgesia to D11 bilaterally, from which point the hypalgesia gradually faded to D8. Vibration could not be detected on the right to the rib cage and was lost in the foot and ankle on the left. Beevor's sign was positive (on raising the head off the bed, the umbilicus ascended cephalad). The deep tendon reflexes were increased in both lower extremities. Babinski's sign was present bilaterally. Ankle clonus could be elicited on the right.

Myelogram revealed block of the spinal canal at D9. The spinal fluid protein was 71 mg. per cent.

At operation an extradural lipoma was discovered and removed. The patient is again walking and improving rapidly.

Warning against too ready use of the diagnosis of multiple sclerosis is appearing more frequently in the literature. Oberhill, Smith and Bucy[2] advise: "Any patient over 35 years of age who for the first time develops symptoms of what might be multiple sclerosis should be investigated for tumor." Rosner[3] finds other conditions closely simulating multiple sclerosis, but more amenable to treatment, to be at least as numerous as multiple sclerosis itself. Fay and Wildebrush[4] report twenty-one cases classified for many years by recognized authorities as instances of multiple sclerosis, which warranted exploratory laminectomy.

Comment. While pain is often the first symptom of intraspinal tumor, as pointed out by Elsberg,[5] it may also be entirely absent depending on whether the position of the mass causes irritation of the sensory roots. Oberhill, Smith and Bucy[2] list as one of the common errors that "Pain and sensory change . . . are sometimes considered essential to a diagnosis." They state emphatically that "This is not the case. Space occupying lesions lying anterior to the spinal cord are particularly likely to produce only motor symptoms without sensory alterations and thus lead to confusion with multiple sclerosis."

As for remission, experience shows, especially with extradural tumors, that the course of the disease is not always steadily downhill but may, for a time, *regress toward normal.* Furtado

and Marquis[6] report the case of a woman with a spinal teratoma and paraplegia who enjoyed periods of complete recovery several times over a span of twent years.

CASE II. F. M., a thirty-two year old, slightly obese white woman, suffered a fall in August, 1949. Immediately thereafter she experienced difficulty in walking. She suffered no pain but described her feet as feeling heavy and numb, more pronounced on the right. Her gait was staggering and her speech was noticed to be slurred. Within a few weeks she began to suffer from constipation and, later, from a tendency toward urinary incontinence. In spite of medical care the condition progressed and she was finally hospitalized in November, 1949.

At this time spastic paraplegia of the lower extremities was noted, more marked on the right. She had become completely unable to walk. Sensory loss was described as patchy, changing from one examination to the next, including vibration, pain and temperature at approximately L1. The deep tendon reflexes of the lower extremities were described as increased and there was a Babinski sign on the right.

A spinal tap was done, showing the fluid to be under normal pressure. The Wassermann test was negative, the cells were not remarkable and the gold curve was within normal limits. The protein, however, was 220 mg. per cent. X-rays of the spine showed no erosion and no widening of the interpeduncular spaces. At first, when myelography was done, it was deemed necessary to run the pantopaque up to the low dorsal region only, since the lower extremities alone were involved. Myelography was reported as negative. The fallacy here is apparent to any neurologist, as extramedullary tumors are often associated with a rising level of sensory loss, gradually climbing to the level of the lesion;[7] also, "leg weakness is usually the first sign of motor involvement due to . . . tumors in the thoracic region (just) below the cervical cord level," according to Alpers.[8]

The patient was now seen by an orthopedist who, wisely, was unsatisfied with the diagnosis, and neurosurgical consultation was called. At this time the patient again exhibited many signs suggestive of multiple sclerosis. Her attitude could now be interpreted as mildly euphoric, the mood perhaps attributable to partial remission. She could again walk with assistance and her bladder control was improved. Her speech was still slurred and there was a sustained nystagmus on looking to the right. Otherwise her signs were confined to the lower extremities.

FIG. 1. Extradural meningioma simulating multiple sclerosis.

There was marked ataxia of gait. Muscular weakness of both lower extremities was present, much more marked on the right. The deep tendon reflexes were markedly exaggerated on the right, and ankle clonus and Babinski's sign were also present on this side. The superficial (abdominal) reflexes were absent. There was a definite sensory level to pinprick and cold on the left up to approximately L1. Position and vibratory sense was poor in both lower extremities. Touch was slightly diminished on both sides up to about L1. This, it was thought, constituted a modified Brown-Séquard syndrome. The upper exremities did not appear to be involved. A graded Queckenstedt test was done now, for the first time, showing almost a complete block. A new myelogram was performed, running the pantopaque higher, and an intraspinal mass was demonstrated at D1. (Fig. 1.)

The spinal fluid protein was now 600 mg.

FIG. 2. Intradural meningioma of posterior spinal cord simulating syphilis.

per cent. Just before operation on December 16, 1949, Horner's syndrome appeared on the right. It was only then, too, that a relative volunteered that the speech defect had existed since childhood.

At surgery an extradural meningioma was found which was wrapped three-quarters around the spinal cord, only the left lateral aspect remaining free. The tumor had insinuated itself on the right into the root canals of C7, C8 and D1. It was possible to remove it completely along with a small section of the dura to which it was attached. Two months later the patient had completely recovered except for persistent Horner's syndrome.

"*Spinal Syphilis*"

CASE III. M. I., a fifty-six year old white woman, complained of progressive impairment of the function of the lower limbs. She walked into the office with the help of two canes, watching her feet and unable to stand if she lifted her eyes from the floor—the typical station of tabes.

For three years she had noticed a dull aching pain in the lumbar area, with pain radiating to the right leg at first. One year before examination she was lifting a heavy object and noticed a snapping sensation in the lumbar area, after which she was completely incapacitated for a short time. She had had a history of urinary incontinence for a period of four or five months. This had cleared up.

X-rays of the lumbar spine had shown generalized rarefaction of the lumbar vertebrae. She, evidently, had not had a spinal tap. She had had treatment for over a year for central nervous system syphilis. More recently she had had treatment for arthritis and neuralgia.

Neurologic findings, which were positive, were loss of vibration and position sense and, to a lesser extent, pinprick over both lower extremities, more marked on the right. The deep tendon reflexes were only slightly increased in the legs, but a Babinski sign was present bilaterally. There was a great deal of impairment of both the sensation and the motor control. Actual motor weakness appeared to be minimal.

A myelogram was done which showed a definite block between the seventh and the eighth thoracic bodies. (Fig. 2.) Pantopaque was then injected by way of the cisterna magna and, again, the block was confirmed at this point.

There was a nodule in the thyroid and, for a while, it was thought that there might be a mass in the pelvis. However, it was not believed that the pelvic examination or the clinical signs indicated metastatic malignancy.

The total protein of the spinal fluid was 366 mg. per cent; the cell count was zero; the Wassermann test was negative; the colloidal gold was 0001233221. The blood count showed 13.5 gm. per cent of hemoglobin but no evidence of pernicious anemia.

The patient, therefore, was operated upon and an intradural meningioma was discovered which had so compressed the cord that it was down to the thickness of a ribbon. The meningioma was removed.

Comment. This case might more logically have been mistaken for combined sclerosis than tabes dorsalis since the reflexes were not depressed. Bailey and Craig[1] note such a possibility. No doubt the striking first impression (of a patient who was unable to stand unless looking at the feet) suggested her first physician's course of treatment. The danger of diagnosis by rule-of-thumb and the fallacy of not following every lead in serious impairment of the locomotor apparatus is illustrated.

"Kidney Trouble"

CASE IV. F. E., a forty-two year old white woman, complained of failure of the bladder to empty of six years' duration. She first noted pain in the lumbar region at the age of seventeen years. She suffered a urinary infection soon after onset, which tended to persist in spite of treatment; and when she finally became unable to void, her difficulty was ascribed to its prolonged effect.

At the time of examination it was necessary for her to catheterize herself daily. Her right calf had begun to be numb several weeks before examination and the "nagging backache" persisted but was attributed, in the manner of the radio advertisements, to "her kidneys." During this period of her life she had had a thyroidectomy, a hysterectomy and a salpingo-oophorectomy, but never a spinal x-ray or tap. Finally a new doctor ordered an x-ray of the spine, which revealed erosion of the pedicles at D10 to L2.

A spinal tap now revealed a spinal fluid protein of 200 mg. per cent. At operation an ependymoma of the filum terminale was discovered.

Comment. Elsberg[5] warned many years ago that "if the tumor begins in a part of the cord which is the center for important reflexes or special function, these may be disturbed early in the disease. Thus, bladder disturbances may be the first symptoms of a growth in the lumbar cord."

Poliomyelitis

CASE V. M. E., a two year old Negro female, was admitted to the hospital because of difficulty in walking and some slight fever. The child had been restless and somewhat nervous for the preceding two weeks. Six days before admission she complained of stiffness and pain in the legs. A temperature of 101.6°F. was found and the child vomited several times. She was referred to the hospital as having a possible case of poliomyelitis. Her mother stated that at that time, after a good night's sleep the night before, the patient got out of bed and found she could not stand. That afternoon, her legs hurt her; they appeared stiff and she could stand only with assistance. She had been constipated for two weeks.

On admission stiffness of the neck and the spine, weakness of the lower extremities and active knee jerks were noted. Spinal tap showed the following: white blood cells, 20; polymorphonuclears, 100 per cent; total protein, 304 mg. per cent; sugar, 80 mg. per cent. Diagnosis of acute anterior poliomyelitis was made.

The next day there was marked nuchal rigidity and tenderness of the left calf and thigh. Hot packs were ordered to the neck, the back and the lower extremities, and the pain improved. A mass of about the size of a half-dollar was noted to the right of the dorsal spine; it was firm and slightly movable, and not tender to palpation. Almost one month after the onset there was complete flaccid paralysis of both lower extremities. X-rays of the spine were taken one week after admission and showed marked increase in the density of the eleventh dorsal vertebra. The pedicles of D10 and D11 on the left appeared very indistinct.

Repeat spinal tap was not done until two months after admission, at which time the leukocytes were 6 and the total protein was 1,835 mg. per cent; xanthochromia was present. It was at this juncture that neurosurgical consultation was called. A definite sensory level bilaterally at about D10 was noted. A myelogram was ordered but because of technical difficulties was not completed until two weeks later, at which time a complete block at the level of L2 was demonstrated.

In summary, this is a case of a two year old girl who suffered from premonitory symptoms for a period of two weeks and awoke one morning with marked weakness of the legs together with stiffness of the neck and the spine. This soon progressed to complete paralysis of both lower extremities and urinary incontinence. Spinal taps revealed a rapidly increasing spinal fluid protein which finally reached 4,000 mg. per cent two months later. Neurologic examination showed a sensory level at D10 and a myelogram revealed an intraspinal space-taking lesion.

Comment. It is to be noted that a diagnosis of poliomyelitis was made in this case on the seasonal occurrence, the age of the patient and the sudden onset. The fact that the reflexes were hyperactive was, evidently, regarded as an "atypical" manifestation. Constipation, which according to Elsberg[9] may be one of the first symptoms (of spinal cord tumor) to appear, was underestimated. There was no record of an original painstaking sensory exami-

nation, but it would be logical to suppose that some sensory change may have been present since a well defined sensory level was found two months after onset. This case illustrates that in the presence of atypical findings there is no substitute for an exacting, aggressive and complete neurologic examination.

Peripheral Neuritis

CASE VI. A forty-seven year old white man was admitted through the emergency room because he "could not control his feet" and had fallen on two occasions. He complained of back pain, pain in both knees and diminished sensation in the legs. (He found he could touch his knees together and was not aware of it.) He had headache and anorexia. He had been living in a rooming house, cooking for himself, drinking moderately and evidently taking an inadequate diet. Examination showed "stocking" anesthesia to sharp and dull up to the knees, and grossly diminished vibration and position sense in both feet. Motor weakness was more marked peripherally than proximally. There was no bladder involvement.

The first impression was peripheral neuritis. However, in this case an alert internist almost immediately had x-rays taken of the chest and the spine. The former showed bronchiogenic carcinoma; the latter, erosion of the pedicles of D12 and L1. A myelogram showed a large filling defect.

Comment. This patient's findings are a good illustration of the medical adage that "there is more harm done by not looking than not knowing." Here a good look (by means of x-ray) established the diagnosis.

REMARKS

That the manifestation of intraspinal tumors is protean is well illustrated by these cases encountered within a three-year period in a new practice of neurosurgery. There were others not described, as for example, a case of "meningitis" which turned out to be extradural abscess, ruptured disc, loosening of the odontoid, etc. The diagnostician's acumen is put to the test; the similitude to other diseases would be discouraging if it were not that in each case there was a clue to the diagnosis which, if duly noted and properly evaluated, would lead an aggressive, determined examiner to further search and eventual success.

Several lessons are well illustrated. First, there is no substitute for a painstaking neurologic examination in every case of nervous system disease, to wit: the "poliomyelitis" case had sensory loss and increased deep tendon reflexes; the second case of "multiple sclerosis" had a Brown-Séquard syndrome. Second, with the x-ray and myelogram as recommended by Horrax[10] now so readily at hand in most communities, many diagnoses can be made merely by "looking" with the help of these media. Third, rule-of-thumb in neurologic diseases is dangerous. Normal dynamics, only slightly increased protein and remissions are found,[11,12] especially when a benign intraspinal tumor is present.

Missing the diagnosis of intraspinal tumor is a medical tragedy, for the majority of such tumors in adults are benign and removable in their early stages, and the patient is often restored completely.[13]

CONCLUSION

Six cases of intraspinal tumor simulating other diseases are presented. In each case there was a clear clue to the diagnosis by examination or x-ray.

Delay or failure of diagnosis of intraspinal tumor is regrettable since most of these tumors are benign and removable in the early stages, with probable restoration of function.

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Laennec's cirrhosis of the liver and posthepatitis cirrhosis are two of the most common intrahepatic conditions producing portal hypertension. Extrahepatic block is caused by Banti's disease and other factors. Direct end-to-side anastomosis is the best shunt operation between portal vein and vena cava but one must be sure the portal vein is not thrombosed. If it is, a lienorenal anastomosis is performed later, through another incision. The mortality rate is less than 7 per cent and the postoperative results are good. Portal hypertension is reduced, hemorrhages from esophageal varices are greatly lessened and liver function is unimpaired by this complete diversion of the portal blood. (*Richard A. Leonardo, M.D.*)

Experiences with the Use of 10 Per cent Aqueous Phenol for Chemical Sympathectomy

PRELIMINARY REPORT

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CHEMICAL sympathectomy signifies the interruption by chemical means of portions of the sympathetic trunk with its included ganglia. These drugs are introduced into or near the location of the trunk by paravertebral injection.

Before proceeding with the main thesis of this paper, we will review some pertinent aspects of the problem which this proposed treatment aims to help. Vascular disorders of the lower extremities (particularly of the occlusive arterial variety) constitute a group of serious diseases affecting an undetermined yet considerable portion of the population today. Moreover, the incidence of such cases will undoubtedly show a substantial rise in the immediate future. This is due not only to the increasingly greater number of people achieving middle and old age, but also possibly to the present day almost universal addiction to tobacco. Existing conservative methods of therapy usually employed are far from satisfactory and, with few exceptions, are uniformly without success in the old, chronic arteriosclerotic type of patient, particularly those with complications such as ulcer or gangrene. In spite of the existence of an enormous literature on this subject, the unsolved problem of definitive therapy still remains a problem which can profitably stand further investigation. At this point the writers must make unmistakably clear their understanding that for obliterative arteriosclerotic disease of the legs (as for other manifestations of arteriosclerosis) no royal road to complete cure exists. The damage to the vessels is generally too far advanced for so favorable an issue. In these cases the physician must be content with partial amelioration of symptoms, the relief of complications and, most important, the saving of limbs.

Integral to each case of peripheral vascular disease is the phenomenon of vasospasm. This factor is generally conceded to be of minor degree and import in the more numerous, sclerotic older age group, and more pronounced in the non-sclerotic, less numerous younger group (Raynaud's, Buerger's disease). The results of our treatment are directly proportional to the amount of spasm involved in the particular case and, more important, to the degree that it can be permanently relieved. Therefore, in our investigation of such cases the most important fact to be determined after the diagnosis has been established is the existence of vasospasm. Many methods have been employed for this determination. Winsor[1] and other investigators,[2–5] after testing a variety of methods (spinal anesthesia, caudal anesthesia, etamon chloride®, priscoline® by injection, remote heat and paravertebral block of the sympathetic trunk with procaine), categorically state that by far the most effective warming and vasodilatation was obtained with the use of paravertebral procaine block. This finding is an important one. It clearly indicates the channels along which our therapeutic efforts should be directed.

Other studies of a confirmatory nature, such as palpation of pulsations of main arteries, oscillometric studies and skin temperature readings, are all important in the work-up of these patients but are not always conclusive determinants of response to therapy.

We will consider some of the present day therapies employed for these ailments. Because of the largely irreversible and advanced nature of the vascular disease encountered, the diffi-

culties and limitations of all forms of therapy will be considerable. Since tissue ischemia is the underlying factor of the entire condition, our therapeutic efforts must at all costs endeavor to improve blood supply to the affected limb. It must be emphasized that even a small but sustained increase in blood supply to a precarious limb may mean the difference between arresting a progressive infection which is jeopardizing the limb, and losing the limb and often the life. In many cases this increase need be no more than is represented by a 1 or 2 degree rise in skin temperature of the toes.

Conservative management along hygienic and medicinal lines constitutes the therapeutic approach most commonly adopted. The panorama of oral medicaments, parenteral agents and mechanical contrivances is extensive and varied; yet these measures all too often fail to engage the problem in a precise, fundamental manner, i.e., to effect a sustained increase in blood supply to a particular portion of the anatomy. Allen, Barker and Hines,[6] and DeCamp,[7] after discussing their experience with most known drugs and apparatus which are commonly employed, conclude that they are all extremely limited in their usefulness. In the words of DeBakey, "these measures . . . transitory . . . none constantly effective." There are a few exceptions: (1) Linton[8] expresses a specific preference for intermittent venous compression in cases of acute vascular occlusion. He believes that combined with anticoagulants it is the treatment of choice for acute thrombotic closure of the main vessels of the leg in arteriosclerotics. (2) Injections of tissue extract are often effective in claudication.[9] However, in most cases its effectiveness diminishes and disappears after a few weeks to months. (3) The value of antibiotics in infective complications is unquestioned. (4) The tonic and vasodilatory effects of alcohol should not be overlooked, nor for that matter overestimated.

Treatment by resection of the lumbar sympathetic chain including ganglia 1, 2 and 3 (usually only the second and third are removed) is often employed and has had many fair to good results. In selected cases improvement results in 30 to 50 per cent of those patients operated upon.[10,11,20] The treatment rests on sound physiologic grounds and has much to commend it in specific instances. However, there is much disagreement among authorities about the indications and contraindications for the operation, i.e., selection of cases.

The conservative school restricts the operation to good risk patients under the age of sixty who show a substantial rise of skin temperature of the extremity following paravertebral procaine block of the sympathetic trunk.[12–16]

The radical school believes that the response to blocks is not conclusive. They advocate surgical sympathectomy for practically all such patients who can undergo the operation when there is no improvement after a trial of conservative management.[17–20] Many of this group practice sympathectomy before a contemplated amputation for the purpose of securing better stump healing and to prevent a possible stump neuralgia.[18,20]

However, the results of sympathectomy are far from uniformly satisfactory. Even where a good result is anticipated (because of a previously determined favorable procaine block test), the outcome all too often is disappointing. The reasons for this are multiple, and the critics of sympathectomy stress the following: (1) The difficulty of removing the first lumbar ganglion via the usual lumbar extraperitoneal approach is a real one and is acknowledged by all operators. (2) The elusive strand of nerve may be missed altogether, even by an experienced operator. (3) The nature of the pathologic condition in this disease is too often largely irreversible.

Having reviewed the medical and surgical approach to this problem, we now proceed to a third method of treatment, namely, sympathectomy by chemical means.

Attempts to interrupt portions of the sympathetic trunk by chemical means introduced paravertebrally have engaged the serious efforts of outstanding men for the past three decades.[22–29] Impressed by the enormous saving to the patient (both in life and in comfort) who might by this means be spared a severe surgical ordeal, and encouraged by numerous reports in the literature both here and abroad of successful clinical application of such methods, the idea at one time attracted considerable attention. Undoubtedly, the apparent simplicity of a paravertebral injection as compared with an open surgical procedure proved to be no unimportant factor in serving to intrigue the interest of many. Unfortunately, this fact also served to beguile many unwary practitioners

who often got into serious difficulties, with the result that the procedure fell largely into disrepute. It should be noted, however, that numerous workers for many years have successfully utilized 100 per cent alcohol for prolonged and even permanent interruption of the sympathetic trunk in scores of reported cases of peripheral vascular disease.[27,29–34] These men were very skillful practitioners who had developed their technic by prolonged study to a high degree of perfection. Thus over the past thirty years chemical sympathectomy has experienced fluctuating fortunes. It varied from enthusiastic acceptance at times by a small group to almost complete rejection and virtual abandonment at other times. Yet despite the disdain of many orthodox surgeons, the indifference of most physicians and the times of periodic oblivion, the concept continues to manifest considerable vitality and to attract considerable work and discussion.

There are several valid reasons for this wavering and somewhat non-enduring acceptance of non-surgical methods of sympathectomy. The more important ones are: (1) Safe drugs like procaine are evanescent in duration, and therefore are useless where a prolonged effect is desired. Such prolonged effect is almost always necessary. (2) Conversely, agents which are capable of producing a prolonged effect (100 per cent alcohol) are not safe. (3) Placing of the needle, even by an expert, is not exact. The effects, therefore, are often uneven and unpredictable. (However, one can always reinject a patient a lot easier than reoperate.) (4) The apparent simplicity of the method gives the operator a false assurance of safety. Hence many accidents may occur through faulty injection by men with insufficient interest and experience. (5) Even with the best technic the method can hardly be as thorough as open operation in which the trunk is resected under direct vision. The last is admittedly true. In our opinion, the preferred procedure in good risk patients with good life expectancy should be open operation. This operation should include the first lumbar ganglion. If necessary, the approach should be as recommended by Smithwick through the bed of the twelfth rib.

However, we are chiefly concerned with the patient past the age of sixty who is suffering from arteriosclerotic vascular disease of the legs and who is afflicted more often than not with other chronic degenerative disorders such as diabetes, heart disease and hypertension. Do we want to subject such patients with an average life expectancy of perhaps three years[21] to the risk of radical surgery? On the other hand, can we rest content with present day conservative methods of treatment?

Are more desirable alternatives available? In the past, as was previously mentioned, 100 per cent alcohol was the principal agent employed for prolonged blocking of the sympathetic trunk. The use of alcohol, however, even with good technic, was often followed by painful neuritis and muscular palsy. At all times, therefore, the chief obstacle to advances in this field has been the lack of a safe chemical agent for injection.

The history of the application of phenol for nerve block is at least twenty-five years old.[24,35–37] It was Mandl[24] who demonstrated that 6 per cent phenol had the unique property of causing the selective destruction of ganglionic cells as contrasted with but a slight irritative effect upon surrounding tissues. In 1947 Haxton,[38] after trying 6 per cent aqueous phenol for paravertebral sympathetic block in peripheral vascular disease with somewhat uneven results, discovered that by increasing the strength of the phenol to 10 per cent he could secure fairly consistent effective and prolonged interruption of the lumbar sympathetic trunk. Three years later he reported a most impressive series of 200 cases of peripheral vascular disease of the lower extremities which had been so treated, namely, the injection of 10 cc. of 10 per cent aqueous phenol paravertebrally into or near the lumbar sympathetic trunk. The over-all amount of benefit obtained was as follows: (1) 50 per cent improvement of swelling in cases of chronic venous insufficiency; (2) 65 per cent improvement in symptoms of claudication; (3) 80 per cent relief of pain, ulceration and digital gangrene in arteriosclerosis obliterans.

The duration of effects lasted up to two years. The injection can always be repeated when the effects wear off. Most important, there were few serious side effects or undesirable sequelae. A report of such magnitude upon so frustrating a therapeutic problem deserves, and will no doubt receive, considerable notice.

The aforementioned findings were confirmed in their entirety by Boyd[39,40] a short time later.

The good results and the absence of serious sequelae were especially noted. Both Boyd and Haxton emphasize that in selected cases the procedure of choice for them is open operation. The previously mentioned method of treatment was reserved exclusively for cases in which patients were too old or sick to justify surgery.

The authors, impressed by the large number of cases studied and the uniformity of good results obtained without serious reactions or undesirable sequelae, and being aware of the widespread interest in this problem, decided to repeat the procedure in similar cases to determine if such results could be duplicated. The following constitutes a preliminary report on our experiences with 10 per cent aqueous phenol. The work was conducted over a period of one year on the general surgical service of a large city hospital. Eleven patients (seventeen limbs) were treated. Although the series was not a large one, each case proved highly instructive.

We had but one criterion for selection of the subject, namely, that he be one who had little or no other therapeutic alternative open to him. Contrary to case selection for open operation and other treatment, we did not look for favorable cases. Each case selected had reached what might be termed a therapeutic impasse. The local and often the general prognosis was an unfavorable one.

With few exceptions all patients were of advanced age (sixty-five to seventy-five years). Almost all had a history of peripheral vascular disease of well over ten years. They were all either bedridden or almost entirely confined to the immediate environs of the home. With few exceptions they had all received for years a large variety of oral and/or parenteral medication for their complaints without benefit. Most of them were afflicted with serious medical complications of long standing (hypertension, coronary disease and diabetes). Coming from the indigent class, those who were ambulant were habituated to bi-weekly clinic visits; and although they hardly ever received any relief for their painful legs, they were content to settle for a little sympathy and placebo medication. Mentally, one would be obliged to describe most of them as somewhat deteriorated. This is a very important point since in follow-up interviews these patients often could see no causal relationship with a particular treatment and improvement.* Few understood the distinction between improvement and complete cure. This last difficulty was not a small one. It was only after careful interrogation that specific details could be extracted from them. We found that we could get more reliable follow-up reports from friends and relatives than from the patients themselves. This was seldom a difficulty with our private patients. They seemed better oriented and were more able to evaluate degree of improvement.

One patient was suffering from advanced mummified gangrene of both feet, and two patients had impending gangrene from acute thrombosis of the superficial femoral artery. There was one old case of milk leg (chronic thrombophlebitis), and one case of amputation stump neuralgia, which had resisted all former therapy, in a severe diabetic of many years' standing. There was one case of ulceration of the toe in a diabetic. The remaining were cases of elderly arteriosclerotics who had pain at rest and severe claudication on walking less than half a block. These cases received the conventional work-up of blood, urine, electrocardiogram, oscillometric study of the legs and skin temperature response to paravertebral procaine. In most cases oscillometric readings were no more than a flicker below the knee and zero above the ankle, and the skin temperature response to procaine block was hardly measurable. In one case no femoral pulsation was palpable. However, we injected them all, ignoring entirely response to procaine blocks or absence of oscillometric readings. We believed that these patients had little to lose in view of the innocuous character of the treatment. We also believed that we had little else to offer them in the way of treatment except ultimate amputation.

COMMENTS

Case I. This was our first case. The patient suffered a severe neuritic reaction in both thighs and severe pain in his back. This lasted four months. Today, with our technic much improved, this case should yield a superior result without severe neuritic reaction (Table 1.)

* One patient (Case III) attributed his improvement to the drugs in the clinic which were "starting to work since the injection." Another man (Case VIII) said his improvement was due to the Swedish massages he was taking.

TABLE 1

SUMMARY OF CASES IN WHICH 10 PER CENT AQUEOUS PHENOL WAS USED

Case and Hospital No., Patient Sex and Age	Dates of Injection	Symptoms and Diagnosis	Response to Procaine	Result with Phenol
I (No. 140028), N. M., male, 68	5/11/50 5/17/50	Diabetes, arteriosclerosis obliterans; burning pains and severe claudication (½ block) both legs; resting cramps	Slight	Good; rise of skin temperature 76°–81°F. right, 76°–86°F. left; resting cramps relieved; could now walk 1½ blocks; benefit maintained 1 yr. then fell off somewhat
II (No. 138710), E. M., male, 67	5/25/50 5/27/50	Arteriosclerosis obliterans; claudication at rest; could do no walking and was an invalid many years; ulcers of dorsum of both legs	None	No change
III (No. 146585), S. F., male, 66	4/8/51 4/13/51	Arteriosclerosis obliterans, coronary sclerosis with angina, hypertension; invalid 20 yr.; claudication (¼ block) and pain at rest	Questionable	Good; marked warming of both legs; could walk 2 to 3 blocks; resting cramps and night cramps relieved; maintained at last follow-up (6 mo.); reaction 1 side
IV (No. 146219), W. G., male, 42	4/10/51	Arteriosclerosis obliterans; severe diabetic 21 yr.; amputation left leg below knee 7 yr. before for gangrene; pain in stump and indolent ulcer for 6 mo., bedridden; femoral pulse absent	None	Immediate result very good; stump became painless and knee joint became more mobile; ulcer began to granulate; result wore off in 4 wk.
V (No. 146103), I. C., male, 62	4/12/51 4/15/51	Arteriosclerosis obliterans, severe diabetes, nephritis, advanced osteoporosis; bilateral mummified gangrene of digits 3 and 4 extending half the distance to ankle; bedridden, cachectic, pain	None	Poor; possible acceleration of gangrenous process
VI (No. 143740), C. C., female, 57	5/10/51	Chronic thrombophlebitis (6 mo. duration); chronic venous insufficiency with dependent edema; leg cold and heavy	Slight	Good; edema receded considerably (80 per cent); leg felt lighter; report on 11/7/51: result maintained
VII (No. 147933), R. O., male, 67	6/13/51	Acute thrombosis right superficial femoral artery; embolectomy tried	None	No change; amputation one week later; primary union of stump
VIII (No. 147480), M. R., male, 77	5/7/51 5/10/51	Arteriosclerosis obliterans; coldness and claudication at rest; walking limit ¼ block	None	Good; marked warming both feet; 7°F. rise in skin temperature of feet, from 84°–91°F.; walking ability increased to 2 blocks, maintained at last follow-up (6 mo.)
IX (No. 148266), J. S., male, 68	7/2/51	Arteriosclerosis obliterans, diabetes, coronary sclerosis; severe pain in right big toe with dime-sized ulcer inside of toe, 4 wk.	Slight	Good; pain in toe subsided 1 day after injection; discharged from hospital 2 days later; follow-up: ulcer healed in 2 wk., patient ambulant, penicillin not given; follow-up 11/5/51: leg felt good; 1°F. warmer than good leg
X (No. 143243), B. F., female, 73	7/11/51 7/20/51	Arteriosclerosis obliterans, diabetes, ulcer dorsum leg; severe claudication both legs and feet, 10 yr.; invalid and bedridden past few years; could walk no more than a few steps	None	Good; warming of both legs; improvement in walking, 1 block; follow-up (4 mo.) 11/5/51: result maintained; ulcer not healed; diabetes poorly managed
XI (No. 147860), R. R., male, 66	7/19/51 7/20/51	Thrombosis of right superficial femoral artery, 2 wk.; cadaveric coldness of right leg from knee down; pain in leg and foot	Good	Immediate result good; right leg and foot became 3°F. warmer until a point a little above toes; pain disappeared except in toes; temperature of foot 6 wk. later but 1°F. above pre-injection level, and pain persisted in toes

Case II. No improvement was obtained. Subsequent surgical sympathectomy yielded no better results and bilateral amputation was necessary.

Case III. The patient suffered a neuritic reaction on his right side, but not on his left. We used a 4 inch needle on the right and a 5 inch needle on the left. The reaction subsided in six weeks.

Case IV. This patient was dramatically relieved of the intense neuralgic pain in his stump for the first time in months. This relief lasted for four weeks and then his pain recurred. Instead of returning for reinjection he elected to return to the surgeon who had originally performed the amputation. He died following operation for reamputation of the leg above the knee.

Case V. This patient was practically moribund. His gangrene was far advanced and bilateral. He was addicted to narcotics. His diabetes was difficult to control. The block was administered with the hope that pain might be relieved, the right leg might be salvaged partially and stump healing might be improved following amputation. He was a poor medical risk and succumbed after amputation of his left leg one week after injection.

Cases VII and XI. These were cases of occlusion of the superficial femoral artery. The first patient (Case VII) came to us at the onset of the condition, and an operation for thrombectomy was attempted, followed six hours later by phenol block. The second patient came to us two weeks after the onset of the condition. No operation was performed; phenol block was administered at two levels (L1, L2), twenty-four hours apart, with considerable improvement of symptoms. However, on September 9, 1951, seven weeks following the block, surgical sympathectomy was performed on the affected side. The immediate effect on the skin temperature of the leg was a rise of 3 degrees (the same as with phenol). Three weeks later, however, the temperature had dropped to preoperative level.

One recent private case (seen January 4, 1954) of embolic closure of the popliteal artery in a seventy-two year old female diabetic with auricular fibrillation which was treated within four hours after onset, manifested almost immediate improvement. The patient made a complete recovery.

TECHNIC OF ADMINISTRATION

The reader is referred to Haxton's or to Boyd's description of the method of administration.[38–40] The salient points are: (1) The solution must be heated and kept hot so that no phenol crystals remain undissolved. (2) The needle must be long enough, not less than 5 inches, 21 or 20 gauge, short bevel. (3) The point of injection must be far from the midspinous line, about ½ inch below the tip of the twelfth rib. (4) The angle of the needle should be about 30 degrees to the sagittal plane of the back. (5) The point of the needle must be placed in front (not alongside) of the second lumbar vertebra. (6) Ten to fifteen cubic centimeters of the solution are slowly injected into the loose retroperitoneal tissues opposite the body of the second lumbar vertebra.

Dangers. It must be emphasized that 10 per cent phenol in the wrong place can be very irritating. The operator must endeavor to follow a careful technic to avoid such an occurrence.

We encountered serious painful sequelae in our first patient injected. This ensued one week after injection and consisted of severe backache and neuritic pains in both thighs. This was due to psoas muscle infiltration and irritation of the genitofemoral and anterior femoral nerves. It lasted well over four months. We had one other neuritic reaction on one side only (Case III), which lasted six weeks. This was caused by the employment of a needle of insufficient length. The balance of our injections (fifteen) was uneventful, or associated with a slight backache for one to two weeks. The average patient injected was out of bed on the same day.

It is needless to add that constant vigilance must be exercised to avoid injecting phenol either into the spinal canal or into the aorta or inferior vena cava. Such injection might lead to serious consequences.

CONCLUSIONS

Few arbitrary conclusions can be drawn from a series as small as this. However, viewed as a preliminary study based upon work covering a much larger series by two earlier investigators, our results tended largely to confirm their findings and to substantiate their claims, with one or two minor exceptions.

Our experience with 10 per cent phenol in aqueous solution utilized for sympathetic trunk interruption in eleven cases of peripheral vascular disease of the lower extremities which were followed up from six to twelve months would suggest the following conclusions. (Table II.)

TABLE II

RESULTS OF 10 PER CENT AQUEOUS PHENOL INJECTION FOR PERIPHERAL VASCULAR DISEASE

Diagnosis	No. of Cases	No. of Extremities	Clinical Result	
			Improved*	Unimproved
Arteriosclerosis obliterans				
(*a*) With claudication	5	10	4	1
(*b*) With ulceration	1	1	1	0
(*c*) With advanced gangrene, bilateral	1	2	0	1
(*d*) Acute femoral occlusion (and embolectomy)	1	1	0	1
(*e*) Subacute femoral occlusion	1	1	1	0
(*f*) Stump neuralgia	1	1	1	0
Chronic venous insufficiency—old thrombophlebitis	1	1	1	0
Total	11	17	8	3

* Improved means rise of skin temperature of 1° to 7°F. and improved walking ability sustained 6 mo. or more in group (*a*), cure of ulcer in (*b*), considerable relief of pain and rise of temperature of leg in (*e*), disappearance of stump neuralgia for 4 wk. in (*f*), and 80 per cent diminution of swelling in chronic venous insufficiency, with much subjective improvement.

Sustained elevations of skin temperature of the feet and legs from 1 to 7 degrees could be secured in over 75 per cent of patients treated (except the case with advanced gangrene and the one with acute vascular block). This elevation of skin temperature was maintained for six months and over. Such increased blood supply to the subcutaneous tissues and skin should help clear up infections, ulcerations, minor gangrenous processes and eczematoid lesions associated with pain and pruritis in cases of ischemic vascular disease. This was also noted in four private cases not listed herein.

Favorable effects on claudication and resting pains were also noted. These effects, although positive, were not dramatic. The usual benefit consisted of an increase of claudication distance from one-quarter to one-half block before injection to one and one-half to three blocks after injection. The latter was maintained over six months. Resting pains were almost entirely relieved in most of our cases.

There was little exact correlation between skin temperature response to test procaine blocks prior to treatment and the results obtained with phenol injection. Most of our patients were so far advanced in age and in vascular damage that test blocks seldom showed a skin temperature rise of more than 1 degree, generally ½ degree or less. Nonetheless, we secured clinical improvement following phenol injection in most subjects treated. In this respect we differ with Haxton and Boyd, who do not inject phenol unless previous trial procaine block yields at least a 1 degree rise of skin temperature of the toes. We would not restrict the injection solely to cases showing a favorable response to procaine block. We believe that favorable results can be secured in a good percentage of those who do not show such favorable response.

There appeared but little correlation between degree of skin temperature rise and degree of improvement in walking distance. Several patients (Cases I, III and VIII) showed sustained temperature rises of more than 5 degrees after phenol injection; yet their improvement in walking was little more than other patients (Cases IX and X) in whom the temperature rise was no more than 1 degree.

It is justifiable to infer that in a somewhat younger age group presenting disease not quite so advanced, the improvement might be more pronounced (seven private cases not included in this series).

The sustained effects noted in this advanced,

almost senile age group appeared to compare favorably with results secured from surgical sympathectomy. Moreover, two patients (Cases II and XI) underwent surgical sympathectomy some time following chemical sympathectomy, with no further improvement in results beyond that obtained with phenol. Although the method is probably not as thorough nor as permanent as open operation, it may prove well worth a trial in particular cases as noted.

The method, contrary to surgical sympathectomy, may be applicable to cases of acute vascular obstruction. In common with surgical sympathectomy, it is not indicated for cases of mummified gangrene (Case V). It may also be tried (Case VIII) some time after the acute obstructive phase, if the limb has survived, with expectation of some benefit.

The treatment can be administered safely and with minimal discomfort to patients of all age groups even though serious complicating diseases may coexist (which would contraindicate surgical sympathectomy) with reasonable assurance of providing measurable improvement in the majority of patients treated. It must be emphasized that the method is safe and free from painful sequelae only if exacting care is exercised during injection. Otherwise the ensuing neuritis can be as severe as that resulting from alcohol injection. In this important point we differ with Haxton and Boyd.[37–39] They lay little stress on severe neuritis as a possible complication.

SUMMARY

A preliminary study utilizing 10 per cent aqueous phenol for chemical sympathectomy in ten cases of arteriosclerosis obliterans and in one case of chronic thrombophlebitis appears to confirm almost entirely the original findings of Haxton and Boyd, i.e., that effective and sustained interruption of lumbar sympathetic nerve outflow could be achieved in the majority of cases so treated. These results were maintained well over six months, the time of our study except for two cases as noted.

The method is safe and free from serious reactions or painful sequelae when careful technic is practiced.

The comparatively simple procedure entailed and the almost entire absence of side effects should merit further application of this treatment to patients past the age of sixty or to those who are surgically poor risks. It is especially indicated for acute closure of peripheral vessels in advanced arteriosclerotics, in whom it may often prove limb-saving.

Finally, in terms of complete cure, we must all admit the inadequacy of all therapies currently available, including the surgical. It is important that this point be made clear to the patient before definitive therapy is instituted, to avoid subsequent misunderstanding.

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WALTERS and Chance, among others, have shown that vagotomy is an ideal procedure to perform in the treatment of marginal or gastrojejunal ulceration which occasionally follows gastrectomy. Results show that three-fourths are cured and operative mortality is only 1 per cent. It would be higher if one attempted re-resection instead. (*Richard A. Leonardo, M.D.*)

Causalgia

EVIDENCE OF THE EXISTENCE OF CROSSED SENSORY SYMPATHETIC FIBERS

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CAUSALGIA is a symptom complex[1–3,7,8] usually consisting of burning pain, hyperesthesia and atrophy of the involved extremity. The pain may be aggravated by contact, motion of the extremity or emotional excitement. It occurs following incomplete (or, rarely, complete) injury to a peripheral nerve trunk.

Early articles on causalgia considered it a sympathetic vasomotor disturbance. The inconstancy of the vasomotor effects and the constancy of the sensory symptoms,[2,3,7,12] however, suggests that causalgia is a disease which primarily involves the sympathetic sensory fibers[15] and that the vasomotor effects, when they occur, are secondary to the sensory component.[19] This point is reinforced by the fact that alteration of the vasomotor tonus by any method other than sympathetic block or sympathectomy (e.g., by generalized body heating or cooling, priscoline,® intra-arterial adrenalin, etc.) fails to relieve the pain of causalgia.[3,14]

Most causes of causalgia can be relieved or markedly improved by sympathetic denervation[1] of the affected part. Shumaker[2] suggests that the failure of sympathetic block to provide temporary relief is probably sufficient to rule out causalgia as the clinical diagnosis. It is believed that the failure of sympathectomy to relieve the symptoms of causalgia[1] and other sympathetic dystrophies[18] may represent incomplete denervation and that some of these failures may be salvaged by higher sympathectomy.

Anatomically, the sympathetic system consists of both sensory and motor fibers. The afferent fibers do *not* synapse[4,5] within the ganglia but rather appear to travel with the sympathetic trunk like "misplaced" somatic fibers. It has been amply shown[6,9,16,17] that painful stimuli can be transmitted via the sympathetic system.

The anatomic and physiologic distribution of the sympathetic system varies in individuals but, in general, distribution is homolateral in the extremities. There are several anatomic studies[10,11] which demonstrate cross communications between the sympathetic chains in up to 80 per cent of cadavers studied. One report[13] states that crossing of sympathetic rami rarely occurs above the L5 vertebra and concludes that bilateral innervation of limbs via such transverse connections "can be almost certainly excluded." There are, to our knowledge, no *clinical* reports of contralateral sympathetic innervation.

We have, in the past, occasionally believed (as have others[10]) that a specific patient might be interpreted as having bilateral distribution of one or both sympathetic chains. However, the clinical evidence of this bilaterality has not been sufficiently clear-cut to be acceptable as proof. We recently studied a patient in whom we believe the clinical observations are adequate to demonstrate the bilateral distribution of the afferent fibers of the right lumbar sympathetic trunk.

CASE REPORT

This patient was first seen by us in August, 1951. He had suffered a high explosive shell fragment wound to the left buttock in October, 1944, at the age of thirty-two. He had been subjected to multiple operative procedures both while in the Army (removal of shell fragments) and since discharge (left sciatic neuroplasty in 1947, left lumbar sympathectomy in 1948 and left thoracolumbar sympathectomy in 1949). A diagnosis of causalgia of the left lower extremity was made prior to Army discharge. The two sympathectomies and the sciatic neuroplasty were unsuccessful attempts to relieve the symptoms of causalgia.

Our admission history and physical examina-

tion revealed, among other things, a fused left hip, a partial left foot drop, multiple surgical and traumatic scars (consistent with the history), burning pain in the left foot, painful muscular spasms of the left foot and lower leg, hyperesthesia of the lateral surface of the dorsum of the left foot, and atrophy (average 4 cm.) of the left thigh and leg. He wore a foot drop brace. The left foot and leg were warmer and drier than the right.

Sweating tests suggested completeness of sympathetic denervation of the left lower extremity. Spinal anesthesia (to T8) temporarily abolished the pain; a "placebo" spinal anesthetic (with physiologic saline) failed to do so.

Spinothalamic tract cordotomy was advised as the only remaining means of controlling the burning pain, muscular spasms and hyperesthesia. In view of the magnitude and complication rate of this procedure it was thought advisable to postpone the cordotomy to allow the patient to "think about it."

He again presented himself to the hospital on May 14, 1953. In the interim he had been examined by a neurosurgeon and cordotomy had again been recommended. The patient thought that some definitive procedure, no matter what its magnitude, had to be performed to control the pain.

The failure of apparently adequate sympathetic surgery to control this patient's causalgia suggested that perhaps his pain was being mediated by afferent sympathetic innervation to the left leg from the right lumbar chain. A right lumbar sympathetic block was done, with complete temporary relief of the burning pain and hyperesthesia of the left foot. On May 20, 1953, a right lumbar sympathectomy was performed, removing the L2 and L3 ganglia, part of the L1 ganglion and the intervening chain. Postoperatively the burning pain and hyperesthesia were completely absent and did not recur. The spasms were markedly decreased postoperatively and had completely disappeared by the fifth postoperative day. There was no apparent change (as evidenced by the lack of change in temperature, color, sweating, peripheral pulsations, etc.) in the vasomotor status of the extremity.

Personal follow-up has been impossible. The patient has since been admitted (October, 1953) to another Veterans Administration hospital with delirium tremens and with various symptoms unrelated to his causalgia. He is still entirely pain-free with regard to his left leg. He still has the atrophy and foot drop, but the burning pain, spasms and hyperesthesia have apparently not recurred.

COMMENTS

It is our opinion that this case clearly demonstrates two points, namely, that crossed sensory sympathetic innervation does occur, and that causalgia is primarily a *sensory* sympathetic disorder. The second point (that causalgia is primarily sensory) seems to follow from the facts that this patient had persistence of his causalgia in spite of previous, apparently adequate, vasomotor denervation and that a contralateral sympathectomy which had no demonstrable effect on the vasomotor status of the extremity did relieve the causalgia. The first point also seems to follow from the clinical course of this patient.

This case, we believe, definitely establishes the existence of crossed sensory sympathetic innervation; we are convinced of, but cannot yet prove, the similar existence of crossing of the vasomotor elements. It is our belief that serious consideration should be given to contralateral sympathectomy, either in the sensory or vasomotor sympathetic dystrophies, when the result of homolateral sympathectomy is less than might be expected or desired.

We did not see this patient until seven years after the injury, approximately two years after-thoracolumbar sympathectomy. It appeared to us that adequate homolateral sympathectomy had been performed. An additional two years elapsed before the contralateral sympathectomy was performed. The result was immediate and apparently continuing relief of his causalgia symptoms.

CONCLUSIONS

The existence of afferent sympathetic fibers is generally accepted. Apparently these fibers are distributed with the efferent fibers. It is the division of these afferent neurons (rather than any vasomotor effect) which produces the relief of symptoms of causalgia. Some of the few therapeutic failures following sympathectomy in causalgia have been salvaged by higher sympathetic denervation. Perhaps, as in our patient, a contralateral sympathectomy might have relieved the remainder. We believe that contralateral afferent fibers were present in this patient; perhaps they are present

in the other causalgia failure of sympathetic denervation.

SUMMARY

A case is presented in which there were persistent symptoms of causalgia in spite of apparently adequate homolateral sympathectomy. A contralateral sympathectomy resulted in complete and continuing relief of pain.

Two conclusions are drawn from this case: (1) that crossed sympathetic sensory fibers do exist; and (2) that causalgia is primarily a sensory sympathetic dystrophy.

One further hypothesis is drawn from inference, that is, crossed sympathetic vasomotor fibers probably also occur.

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Renal Function after Complete Bilateral Ureteral Obstruction Following Colporrhaphy

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THE occurrence of bilateral ureteral obstruction following surgery is an unusual but grave complication. This cause of anuria occurs most commonly after abdominal hysterectomy, with a higher incidence after the more extensive or difficult procedures.[7b] Following an anterior colporrhaphy, bilateral occlusion has been reported only three times in the English literature.[1,3,57] This paper reports a fourth patient, who also presented a rare opportunity for the study of renal function during a definite complete ureteral block.

CASE REPORT

F. B. (Hospital No. 27163), a fifty-six year old white woman, developed a recurrent third degree cystocele with a moderate vaginal prolapse a few months following vaginal hysterectomy and vaginal plastic operation which was performed two and one-half years before this admission. The large cystocele was repaired with No. 00 chromic catgut on April 1, 1953, at the Mt. Sinai Hospital of Greater Miami. An indwelling urethral catheter drained no urine following the operation. After twelve hours of anuria cystoscopy revealed no ureteral efflux and catheters could be inserted no more than 1 cm. into both ureteral orifices. However, skiodan 20 per cent injected through an acorn-tipped, occlusion catheter outlined sharp kinks of both lower ureters, which were now dilated from the obstruction. (Fig. 1.) The radiopaque iodide could be forced past the kinks only by using a moderate amount of syringe pressure. (Fig. 2.) A plain film ten hours later still showed dye retained in both lower ureters. (Fig. 3.)

The patient remained anuric for another two days although her general condition was good, the only complaint being bilateral lower abdominal pain. Finally after seventy-two hours without urine reaching the bladder, a suprapubic freeing of the right ureter was unsuccessfully attempted even with opening of the bladder, due to dense adhesions; therefore a right nephrostomy was immediately performed. As soon as the nephrostomy tube was inserted into the distended pelvis, it drained urine, 2,000 to 3,000 cc. during each of the next two days. The patient improved rapidly and became ambulatory. The left ureter remained completely occluded since no urine reached the bladder, vagina, rectum or abdominal cavity. After thirteen days an intravenous pyelogram (I.V.P.) was performed with the right nephrostomy tube clamped and amazingly demonstrated function in the completely blocked, hydronephrotic left kidney. (Fig. 4.) On the twenty-third day the patient began to void large amounts of urine presumably from relief of the left ureteral blockage, since the right nephrostomy catheter continued to drain larger amounts of urine. On the twenty-fourth day an I.V.P. with the right nephrostomy tube open showed an increase in the left hydronephrosis. (Fig. 5.) The right ureter was also patent by this time since indigo carmine injected into the nephrostomy tube could be seen through the cytoscope spurting from the right ureteral orifice. Catheters still could not be passed more than 1 cm. bilaterally.

The patient was discharged thirty-six days after the original plastic surgery, voiding all the urine with the nephrostomy tube constantly clamped. On the forty-seventh postoperative day she was readmitted as a service patient with fever, chills, left flank pain and a non-functioning left kidney on I.V.P. A left nephrostomy was performed by another urologist on the fifty-second day but actually this tube never drained urine and was found on x-ray not to be within the left renal pelvis. With antibiotics the patient became afebrile

FIG. 1. The left kink is demonstrated with skiodan injected up the lower one-half of ureter. Radiopaque dye is in bladder from reflux around the occlusion catheter during injection. The base of the bladder and ureter are lower than usual in relation to the symphysis because the patient's legs are in stirrups resulting in rotation of the pelvis. Michel clips are present over the left ileum where a superficial lipoma had also been removed.

and both kidneys revealed good function with some hydronephrosis on I.V.P. Thus this latter episode represented an acute left pyelonephritis which responded to antibiotics.

The nephrostomy tubes were removed on the sixty-eighth day and the patient was discharged seventy-four days after the colporrhaphy with both wounds healed. Since then the patient has had no urinary symptoms and has remained in good health. A follow-up study four months after colporrhaphy revealed a normal urinalysis. An intravenous urogram showed prompt five-minute bilateral visualization with a normal left pelvis and ureter, but a mild right hydronephrosis. (Fig. 6.) After nine months the patient's general condition was excellent.

Comments. The ureters had not been actually caught by the sutures. Yet a complete functional bilateral ureteral obstruction had been produced by the bilateral kinking even though dye could be forced past the kinks with occlusion catheters using a moderate amount of syringe pressure. With this type of injection Hinman and Rose have shown that forces of 150–200 mm. of mercury are easily produced. On the other hand several investigators[26,28,40,50,55,80] after ligating the ureters of sheep and dogs have shown that a maximum renal secretory pressure of 70–90 mm. Hg results after three or four hours of blockage, the pressure decreasing to 30–40 mm. Hg after twelve to twenty-four hours and to 15 mm. Hg after thirty days of obstruction. Human kidneys probably parallel these figures with Walker recording pressures of 35–45 mm. Hg after only twenty-five minutes of occlusion, the same values as in animals after twenty-five minutes of obstruction. Thus in this patient under report, although skiodan could be injected by the kinked areas utilizing a syringe, the renal secretory pressure was not great enough to force urine through the twisted portions of the ureters.

The patient's course proves that a functionally complete obstruction existed. The radiopaque dye forced past the kinks (Figs. 1 and 2) demonstrated the rapid effects of the obstruction since after only twelve hours the ureters were moderately dilated. The ureters were probably of normal caliber before the operation since follow-up films four months later (Fig. 6) showed normal-sized lower ureters. A delayed film taken ten hours after dye injection through the occlusion catheters confirmed the obstruction since dye was still present in the lower ureters. (Fig. 3.) Finally, after three

2 3

FIG. 2. Dye is now injected into right ureter and pelvis with the upper segment of the obstructing kink obscured by the cystoscope. Note dilatation of both ureters and the twisted elongated upper right ureter as the result of a twelve-hour occlusion. The skiodan in the left ureter is blocked at the beginning of the previously visualized kink.

FIG. 3. Radiopaque iodide still present in both lower ureters ten hours later, partly obscured on the right side by intestinal shadows.

days of anuria operation showed a distended right renal pelvis and upper ureter, and the nephrostomy tube drained urine immediately with 2,000 to 3,000 cc. obtained on each of the next two days and thereafter 1,000 to 1,500 cc. each day.

The most amazing aspect of this case is that an I.V.P. after thirteen days of complete left renal blockage showed dye in the distended calices in fifteen minutes and in forty-five minutes in the dilated pelvis. (Fig. 4.) The left ureter was completely occluded as no urine was obtained with an indwelling urethral catheter. When the catheter was removed the patient did not void and subsequent catheterizations obtained no bladder urine. Careful daily examinations revealed no vaginal or rectal leakage of urine. There were no signs of peritoneal irritation from a possible escape of urine intra-abdominally. Experimental work[4,31] and an occasional clinical case[1,29,34,48,69] have shown this latter process to be a possibility.

In reviewing the English literature only eighty-nine episodes of complete bilateral occlusions have been reported following gynecologic surgery (Herman, twenty-four cases; Bland, twenty-five; Fiener, fifteen; Adams, seventeen; Reeves, one; Moore, two; Henderson, one; Rubin, one; Aschner, two; St. Martin, one.)* A careful review of these series revealed only one case in which function was claimed on an I.V.P. in a totally obstructed kidney.[42] However, a close examination of this history indicated that bilateral ureterovaginal fistulas existed and therefore kidney function was present three months later only because the obstruction was partial. Moore also performed an I.V.P. on one patient who was anuric for three days with no evident visual function.

* There are eighteen other instances of incomplete bilateral injuries mentioned in these references. Thirty-one other cases from these papers, as well as five from Prentiss et al., cannot be classified because no data were available.

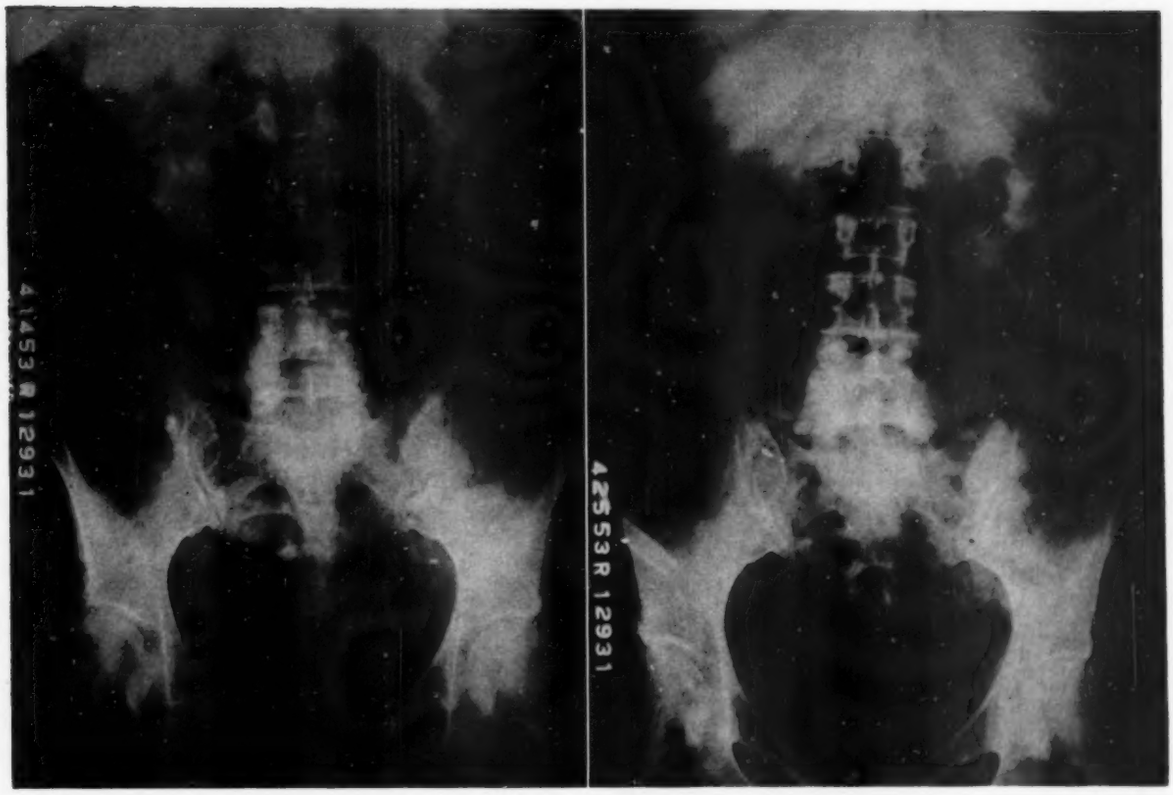

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FIG. 4. On the forty-five-minute film of an I.V.P., with the right nephrostomy tube clamped, dye fills the right calyces, pelvis and ureter down to the obstructing kink. The hydronephrotic left kidney, blocked completely for thirteen days, shows iodide in the calyces and pelvis. The faint shadow in the left lower bony pelvis is probably produced by the intestines since it was present on the plain film.

FIG. 5. Although the patient was voiding for one day before this I.V.P. was taken on the twenty-fourth postoperative day, no radiopaque dye is present in the bladder on this thirty-minute film. The left hydronephrosis is slightly increased. The right nephrostomy tube is open during this examination but dye is visible in the right lower ureter as well as the left.

In addition to the unilateral injuries in the foregoing references, the occurrences of all the other unilateral injuries following pelvic surgery, amounting to 859 cases, were reviewed.[5,8,13,22,23,41,45,49,51,58,60,64,68] Less than one hundred of these were complete occlusions, the exact number being indeterminate because of perfunctory records. In only three of the total occlusions were postoperative urograms recorded, two after the sixth and one after the eighteenth postoperative day, and none showed excretion on the blocked side.[41,62,69] Unquestionably, many other cases of non-functioning kidneys with an I.V.P. following surgical complete blockages have not been deemed important enough to report in journals. Frequently no visualization occurs in other types of lesions even with incomplete obstructions as Woodruff and Wilcox have recorded.

Why then did visualization occur in this patient with a totally blocked kidney and not in the previously mentioned cases? An important contributory factor that was never carried out before was the clamping of the nephrostomy tube during the procedure which maintained high tissue levels of organic iodide; thus more iodide was available for the left kidney to excrete. Another factor in cases such as Woodruff mentions is the marked suppression of urine formation which occurs only in the first one or two days in ureteral injuries of all types.[39] This decrease in excretion in just the first few days may be due to the initial high intrarenal pressure which at this time approximates the glomerular filtration pressure of 40–50 mm. of mercury (glomerular capillary pressure of 65–75 mm. Hg less protein osmotic pressure of 25 mm. Hg). The use of iodides with

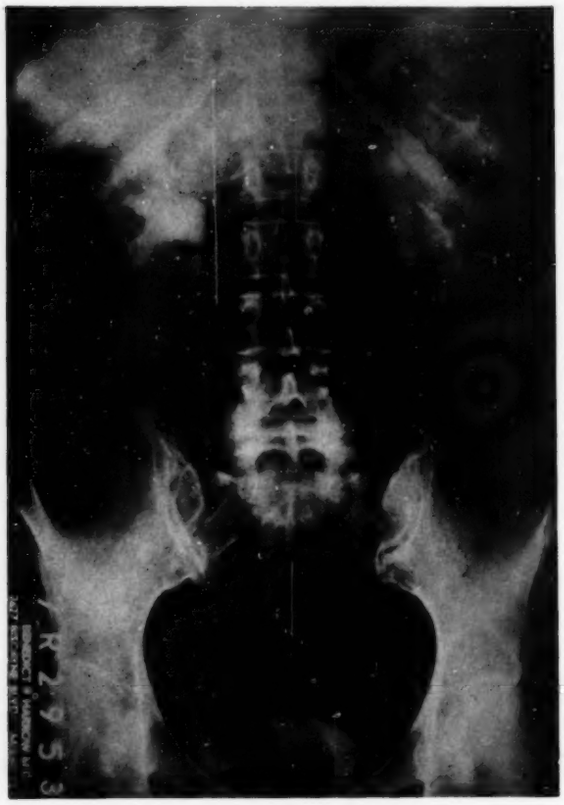

FIG. 6. A urogram (twenty-minute film) four months later reveals normal calyces, pelvis and ureter on the left side with a thick cortex. Some hydronephrosis remains on the right.

different modes of excretion, such as neo-iopax, may be another cause.

RENAL FUNCTION AFTER COMPLETE OBSTRUCTION

It has been proven beyond doubt that renal function continues with total ureteral occlusions. This is the first time, however, that visual proof has been obtained. Scott, Hinman[24,25,28] and others[6,9,18,32] in a variety of vertebrate animals have repeatedly demonstrated that after complete ductal obstructions the glomerular kidney is the only organ that almost invariably undergoes dilatation. The hydronephrosis reaches its maximum in approximately ninety days, depending upon kidney weight, later to be followed by atrophy in about one year. The salivary glands, pancreas and liver undergo immediate atrophy. Unlike partial or intermittant obstruction in which the kidney may attain tremendous proportions, this type of hydronephrosis reaches only a limited size depending upon kidney weight. The mere fact that hydronephrosis develops indicates that some excretion must be continuing. Hydronephrosis is not to be compared with a stagnant pool but rather with a fresh water lake in which water pours in and out.

Studies of urea and sugar concentrations in the urine of obstructed pelves indicates continuous glomerular and tubular activity since the urea concentration remains considerably elevated for several months gradually decreasing toward the blood level while conversely the glucose value gradually increases toward the blood levels.[28] Clinically further proof of renal activity was observed in two anuric patients following bilateral ligation. Indigo carmine was injected intravenously on the second day of anuria, one or two hours preceding nephrostomy. The urine which gushed forth as soon as the pelves were opened was colored blue by the indigo carmine.[15,65] Mc Cahey concluded that renal function did not completely cease in patients with suspected complete block due to calculi, since urograms showed function though urine efflux and indigo carmine did not appear on the affected side cystoscopically. Of course it cannot be proven that these obstructions were complete. In another clinical aspect the well known nephrogram obtained in early complete obstruction in itself indicates continuing excretion.[16,33,63,76,77]

MECHANISM OF HYDRONEPHROSIS

Florence et al. and Wall believed that in total blockades glomerular filtration ceases while tubular secretion continues, thus causing the nephrogram. This theory is disproved by the fact that neo-iopax, which Smith has shown to be excreted almost entirely by filtration, also produces the nephrogram.[74,77] Wilmer believed that glomerular activity stopped since tubular damage in mercury poisoning did not occur if the ureters were ligated. He assumed that mercury was normally excreted by filtration, being influenced by Richard's work. However, the transported mercury is almost totally combined with plasma proteins[52] and therefore cannot be filtered rapidly but probably is secreted by the proximal tubules as an ancillary renal function. Further evidence of this mechanism could be obtained by poisoning aglomerular fish with mercury since tubular damage should result despite lack of filtration. It ap-

pears then, that tubular activity is depressed to a greater degree than the glomerular activity. Additional proof for this is seen in the physiologic osmotic nephrosis. The changes produced in this nephrosis depend upon the osmotic pressures of intratubular substances such as sucrose or glucose brought into the tubular lumens by glomerular filtration. Ligating the ureter does not prevent this lesion[2,79] and indicates that filtration must continue despite the occluded ureter. The marked decrease in the phosphatase in the hydronephrotic kidneys[78] is another indication of the poor tubular function in the blocked organs.

It is obvious that the urine produced in the manner just described in total occlusions must also be continuously reabsorbed. The site of absorption—whether it be the pelvic and calyceal urothelium, pyelovenous reflux, pyelosinous transflow or the collecting tubules—has never been definitely determined.[11] I believe that the postoperative pyelogram (Fig. 4), performed thirteen days after complete obstruction, tends to rule out pyelovenous reflux since the dye must be reabsorbed more slowly than the urine for it to visualize. If the pyelovenous reflux had occurred, the iodide would disappear as rapidly as the urine and not be visible on x-ray. By the same token the pyelosinous transflow of Narath is discarded as the mechanism of absorption. Hinman[26] demonstrated the pyelovenous reflux at low pressures in the obstructed kidney. In his few experiments of prolonged hydronephrosis venous reflux occurred at pressures below that of the secretory forces influencing Hinman's belief that absorption took place by way of the pyelovenous mechanism. However, he recorded limited data in only two or three tests. Furthermore, Fuchs and also Lee Brown demonstrated that in pigs and rabbits, very high pressures amounting to over 100 mm. of Hg were necessary to cause the venous reflux, proving that this reflux could not be the process of absorption in prolonged hydronephrosis. Rolnick and Singer demonstrated early changes in urea and chloride concentrations in blocked pelves which would not have resulted if pyelovenous reflux occurred. They concluded that a differential absorptive process by the epithelium took place. All the data against the absorption by the collecting tubules as a process of the hydronephrosis have been presented before.[26] In conclusion the most likely explanation for the outflow of urine in complete obstructions is the pelvic and calyceal urothelium reabsorption.

OTHER CLINICAL ASPECTS

Clinically Figure 3 illustrates another well known experimental fact that the epithelium of the lower ureter absorbs more slowly than the upper ureter and pelvis.[44] Skiodan which was injected through an acorn-tipped catheter past the kinks had been absorbed from the upper ureters and right renal pelvis in ten hours but had remained in both lower ureters. Figure 2 also demonstrates the rapidity of the dilatation of the ureters, the moderate hydroureters resulting after only twelve hours of occlusion.

This patient's course also illustrated that considerable renal function will return after complete occlusion over a twenty-three-day period since the left kidney had been blocked for that length of time. St. Martin et al. exhibited return of function after nineteen days and Day showed kidney recovery after twenty-three days of complete ureteral ligation. Hinman[28] in his renal counterbalance research in dogs found that a considerable degree of function will return after four weeks of block and even after longer periods if the other kidney is gradually destroyed.

Barney demonstrated re-establishment of the lumen after ureteral catgut ligation. Caulk and Fisher experimentally demonstrated that No. 2 plain catgut ureteral ligatures were never absorbed in less than three weeks in dogs and that six weeks is usually required for a channel to be formed and eight weeks for maximum lumen diameter to appear. In one of Caulk's patients, who underwent bilateral nephrostomies, the ureters did not reopen until the fifty-eighth day. In cases with ureteral kinking an earlier relief may be anticipated. However, this patient demonstrated that even though the ureters are not actually ligated but only kinked in producing a complete obstruction, early operative interference is indicated since the occlusion lasted twenty-three days. As shown in the beginning of this paper, renal secretory pressure is never great, decreasing to very low levels after a few days, and thus represents only a secondary factor in the re-establishment of the lumen. Very rarely only edema and inflammation may obstruct the ureter; yet this anuria may last more than a few days so that urinary diversion also has to be performed.[1,41]

SYMPTOMS AND DIAGNOSIS

In the patient discussed another interesting point is that no flank pain or tenderness developed during the anuric phase, only lower abdominal pain. Other investigators have pointed out that most of the patients with anuria developed no costovertebral angle (C.V.A.) tenderness or pain.[21,48] In this patient left flank pain and C.V.A. tenderness later developed on the tenth postoperative day and lasted one week. Since in total blockades the size of the hydronephrosis is limited (experimentally and clinically as demonstrated by this case, Fig. 5), the kidney will not be palpable except in a thin patient. Because of this fact and the frequent lack of symptoms many unilateral injuries are overlooked.

In diagnosing the cause of anuria, cystoscopy should be performed during the initial ten to twenty-four-hour period. Even though the passage of ureteral catheters are obstructed in the lower ureter, ureterograms with an occlusion catheter must be performed since the catheter may be caught in epithelial folds giving just an impression of ureteral obstruction. In differentiating between bilateral complete obstruction and a rare lower nephron nephrosis with bladder edema so that catheterizing of the ureter is not possible (yet the ureters are patent), an I.V.P. may be invaluable. If the nephrogram is produced or dye appears in the pelves, then an obstructive lesion must exist. Indigo carmine would be of no aid in the diagnosis for it would not appear at the ureteral orifices in either instance.

TREATMENT AND PREVENTION

As to the treatment of anuria due to bilateral ureteral injuries, most authorities (Slutsky, Feiner, Adams, Moore, Aschner, Everett, TeLinde and St. Martin) recommended immediate bilateral nephrostomy, while some urologic surgeons as another alternative suggest intubated ureterostomy with a small Foley or straight urethral catheter.[12,20,36,42,51,53] An intubated ureterostomy through a muscle splitting ureterolithotomy incision at the mid-ureter would be the easiest and least shock-producing operation, and it can be performed bilaterally without the necessity of moving the patient. Vose and Dixey demonstrated that ureterostomy was feasible since the Foley catheter could easily be changed after the fistular tract is established, although further experience with this method is needed. Pyelostomy is not favored because difficulty may be encountered in replacing the catheter, and the patient has to be turned for a bilateral procedure. The earlier writers, Herman, Barney and Bland, performed deligations but recent experience has superceded their opinions. Besides deligation, uretero-ureteral anastomosis, ureteroneovesicostomy, transuretero-ureteral anastomosis, or ureterocolostomy might be necessary but should not be performed as a primary operation until the patient is in good condition. It is generally considered too hazardous to attack initially the obstructed area when careful and often prolonged surgery may be necessary in a patient who has just undergone a major procedure.

An exception might be the cases following vaginal surgery and in this patient under report vaginal deligation was strongly considered. However, this procedure may not be as feasible or successful as it would appear even with ureteral catheters elevating the obstructed area. It was attempted in two of the three previous cases following colporrhaphy. In one, a permanent vesicovaginal fistula resulted and the other was unsuccessful, requiring a subsequent nephrostomy and also producing a ureterovaginal fistula.[1,3] A secondary consideration in vaginal deligation is that a later and more difficult reoperation would be necessary to repair the original vaginal defect. After vaginal hysterectomy the danger of causing bleeding with removal of sutures would also have to be considered.

The operation to relieve the anuria should be performed as early after diagnosis as is possible in spite of the fact that Strauss in his review of the literature found five authentic cases of anuric patients living for twenty-two, twenty-three, twenty-five, twenty-seven and thirty days. Many die in one to two weeks even with proper fluid and electrolyte balance.

Several urologists, surgeons and gynecologists have suggested routine catheterization of ureters before pelvic surgery, but this procedure is performed infrequently. Whenever a difficult operation is anticipated the insertion of ureteral catheters is again strongly recommended. With recurrent cystoceles, especially those associated with a prolapse of the vaginal cuff, redundant ureters may anatomically be more closely associated than usual with the cardinal

ligaments and pelvic fascia used in the repair. Therefore, the ureters can be drawn into kinks by pulling these fascial planes together. If this repair is to be performed vaginally, ureteral catheters should be inserted preoperatively.

SUMMARY

This report is a presentation of the ninetieth patient in the English literature with complete bilateral ureteral obstruction following gynecologic surgery. The patient also represents only the fourth such episode following an anterior colporrhaphy. It is the fourth proven case of complete ureteral obstruction due to kinking[21,68] and is the only reported case in which x-rays demonstrated the kinks. The most interesting information obtained, however, was that visualization of iodide occurred in a kidney completely obstructed for thirteen days. The kidney remained obstructed over a twenty-three-day period, yet follow-up urograms after four months revealed a normal pyelogram. The experimental and clinical data of renal function and the low secretory pressure continuing in hydronephrosis during complete obstruction have been discussed with the conclusion that the main mechanism is glomerular filtration and urothelium reabsorption.

This case emphasized that kinking alone can produce a complete obstruction which may last for several weeks and thus requires early operative interference to relieve the anuria. The rapidity of the hydronephrosis and hydroureter with complete occlusion, the limited extent of the hydronephrosis, the slow reabsorption from the lower segment of the ureter and the lack of flank pain were all demonstrated in this patient.

The use of the occlusion catheter is invaluable in making an accurate diagnosis in bilateral ureteral obstruction. Bilateral nephrostomy or intubated ureterostomy rather than deligation is recommended as the treatment of choice even after vaginal surgery. Insertion of ureteral catheters in secondary colporrhaphy or in anticipated difficult abdominal surgery is strongly advised as a preventative measure.

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Tumors of the Small Intestine

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TUMORS of the small intestine should not be regarded as surgical rarities. The cases reported have so increased that they no longer need be enumerated. While a primary neoplasm of the small bowel is not often encountered, this lesion deserves routine consideration in the differential diagnosis of intestinal distress or bleeding. The following case reports illustrate how careful attention revealed tumors which otherwise would have remained undiscovered.

CASE REPORTS

CASE 1. J. B., a fifty-five year old white man, had been perfectly well until October, 1950, when symptoms of mild prostate infection developed. After a week physical examination was completely normal except for a hard area in the anterior rectal wall, almost 1¾ inches above the prostate. Laboratory findings were as follows: Wassermann and Kline tests were negative. Urine was clear except for a few white blood cells. Hemoglobin was 74 per cent, red blood count 3,920,000 and white blood count 7,400. Cholecystogram and barium examinations of the esophagus, stomach, duodenum and colon were reported as normal. Patient was examined and cystoscoped under anesthesia by a urologist and a surgeon. The opinion stated was that "this mass might well represent an ectopic seminal vesicle but a neoplasm between the bladder and rectum should be ruled out."

No definite advice was given the patient and after waiting three weeks he consulted me. He now felt completely well. His appetite was good, he had no symptoms, bowel movements were normal and no weight loss was noted. The patient was 5 feet, 7 inches tall and weighed 180 pounds. He was hospitalized on November 21, 1950. His blood was type A positive, and the count was the same as before. There was a ¾ inch hard irregular growth in the upper end of the left testicle. On excision it was found to be a spermatocele. The perirectal mass was removed through the anus. It was an irregular, gelatinous structure, 5 by 2.5 cm., lying outside the wall of the rectum. Microscopic diagnosis was "mucinous (colloid) adenocarcinoma which appears to be metastatic in origin." A review was made of the x-ray plates taken one month before which had been interpreted as "negative." It was noted that the fourth part of the duodenum was not clearly seen due to faulty positioning. (Fig. 1.) Therefore, special studies of the small intestine for a possible tumor were requested. The report was as follows: "Duodenal cap is not unusual. There is a dilatation of the duodenum in the region of the jejunal juncture and an irregular filling defect is observed in the proximal jejunal region. The mucosal marking appear irregular and there is a concave deformity in this area suggestive of an intrinsic neoplastic lesion." (Fig. 2.)

The patient was operated upon on December 5, 1950. Just at the ligament of Treitz, there was a 5 by 3 cm. carcinomatous mass on the outside of the jejunum, seeming to have spread from an oval growth within the intestinal lumen. There were a few translucent glands in the thickened mesentery. The duodenum proximal to the growth was dilated to 5 cm. in diameter as was the jejunum for a distance of 12 cm. beyond it. The only distant metastases palpated in the whole abdomen were in the left lobe of the liver which had several hard masses within its substance. The involved intestine (Fig. 3) was excised with 5 cm. of the duodenum and a triangular section of the indurated mesentery extending to the superior mesenteric artery. The procedure was difficult due to patient's obesity and the deep position of the lesion. An end-to-end anastomosis was performed. He had an excellent convalescence and went home on the eleventh postoperative day. Pathologic diagnosis of the specimen was "mucinous, papillary, polypoid and ulcerated adenocarcinoma of the jejunum with involvement of the mesentery. Two lymph nodes

FIG. 1. Case 1. X-ray plate of stomach and duodenum reported as "negative." Because of faulty positioning of the patient the duodenum, and thus the lesion, is not visible.

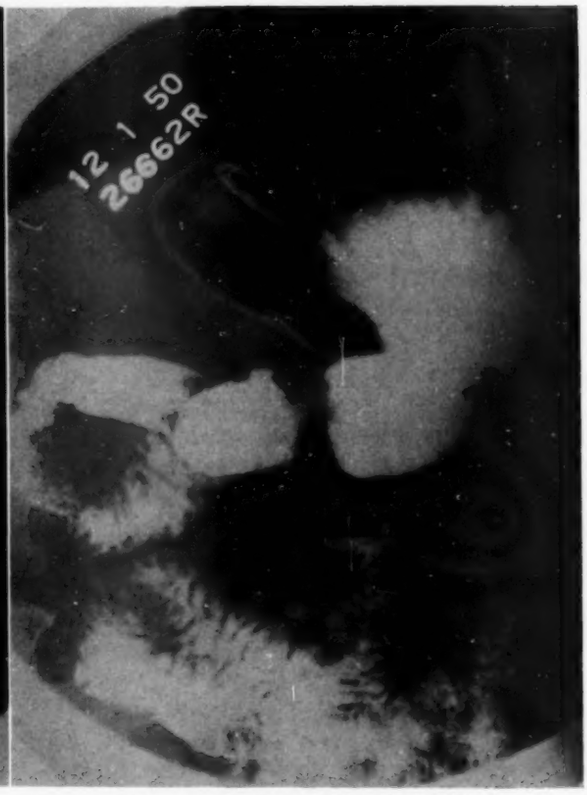

FIG. 2. Same case. Re-examination with better view of upper intestine reveals crescent-shaped deformity of jejunum just distal to ligament of Treitz. The lumen is almost completely compressed due to an intraluminal mass.

found; none show intrinsic tumor metastasis." (Fig. 4.)

The patient remained well until May 1, 1951, at which time cramps of the upper left quadrant of the abdomen developed and some postprandial vomiting occurred. No abdominal mass was felt. There had been a few pounds' weight loss. Hemoglobin was 78 per cent, red blood count 4,090,000 and white blood count 8,500. X-ray diagnosis was "general distention of the small intestine with barium in the ascending colon. Forty-eight hour plates showed some barium in the small intestine and in the colon. This is indicative of partial intestinal obstruction as due to mesenteric glands." The patient was again hospitalized on May 26th because of marked nausea, epigastric cramps and vomiting. Bowel movements were not abnormal. There was a large, hard mass in the anterior rectal wall. Hemoglobin was 81 per cent, red blood count 4,340,000 and white blood count 5,200. The abdomen soon became distended and visible intestinal loops were present. The patient re-entered the hospital on July 6th, at which time he had a large, painless, fixed mass involving the upper two thirds of the left side of the abdomen. He vomited most of his food. Hemoglobin was 71 per cent, red blood count 4,020,000 and white blood count 7,900. The patient died on July 30th. He had lost a total of 40 pounds in weight.

As reported by the pathologist the important points in the autopsy were as follows: abdominal carcinomatosis, extensive, with metastases or direct extension to liver, diaphragm, abdominal and lower left thoracic wall, all omenta and mesenteries, sternum, and the following lymph nodes: periesophageal, perigastric, peripancreatic, lesser omentum, mesentery of small intestine, retroperitoneal. At the site of the enteroenterostomy in the third part of the duodenum, there was recurrence completely around the anastomosis, but the mucosal involvement, which was only 5 to 10 mm. wide, seemed continuous with the external mass in the mesentery. Microscopic

examination revealed the tumor to be, in practically all locations, a fairly well differentiated non-mucinous adenocarcinoma. There were extensive necroses within the tumor and moderate to marked fibrosis. Only in the sections of the perirectal and perisigmoidal tissues and in the walls of the gut in this area was the tumor of the colloid variety, where it was almost exclusively of the mucinous colloid type.

CASE II. V. D., a Negress, forty-eight, had attacks of epigastric distress for four years. At the onset she had only dull aching cramps, but these soon became very painful. The colic occurred about half an hour after meals and she would vomit everything she had ingested. This did not always relieve the pain. During the initial years she was sick only on Tuesday and felt well the rest of the week. Sometimes she vomited only bile-stained fluid. During August, 1951, she passed the only bloody bowel movement she ever had. She was then studied in a university hospital where the final diagnosis was, "psychoneurosis, secondary anemia, and lues." She was treated for peptic ulcer and gallbladder trouble by three other doctors, but this did not help her. When she consulted me in June, 1952, she always carried a toy bucket; and she would appear quite happy as soon as her vomiting was over.

FIG. 3. Case 1. Adenocarcinoma of jejunum, just distal to the ligament of Treitz. The extension outside the intestinal wall is larger than the intraluminal tumor. The duodenal mucosa is seen on the left.

She seemed to be a gay psychoneurotic. She had lost 10 pounds in weight in a year.

Physical examination revealed no abnormality. She was 5 feet 5 inches tall and weighed 142 pounds. Her abdomen was flat and no masses or tenderness were felt. No hyper-

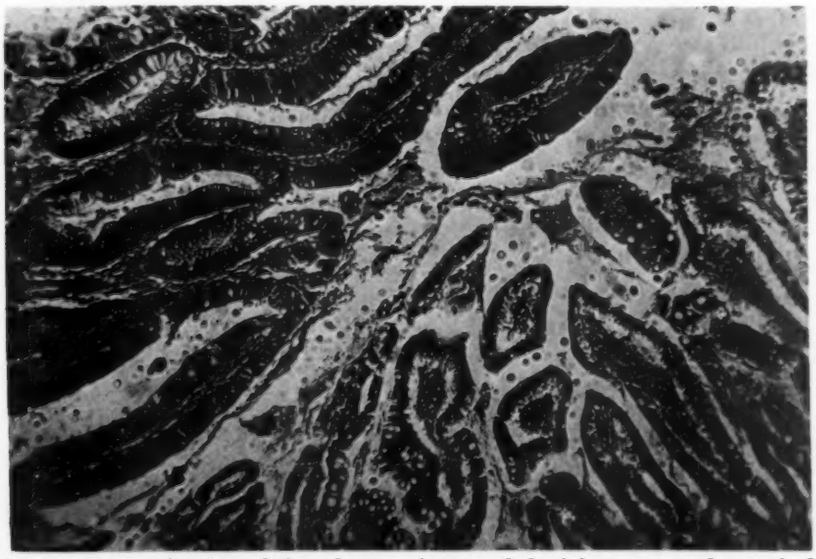

FIG. 4. Case 1. Section of the adenocarcinoma of the jejunum reveals atypical glandular proliferation, loss of polarity of the epithelial cells, stratification of nuclei, bizzare nuclei, fairly frequent mitotic figures and a small amount of mucus production. In the adjacent normal mucosa, on the right, there is a normal intestinal mucous membrane, having a single row of tall columnar cells and basely placed, small, regular nuclei.

FIG. 5. Case 11. X-ray plate of upper intestine reported as "negative." After excision of lipoma just beyond ligament of Treitz, a globular dilatation of the jejunum is recognized in this area. There is widening of the intestinal folds and irregularity of the markings, as seen in an intussusception.

peristalsis was heard. Nothing of note was found on vaginal examination. The blood picture, similar to the one ten months before, was: hemoglobin 72 per cent, red blood count 4,100,000, white blood count 6,800. Intravenous pyelogram, oral cholecystogram and barium studies for possible small intestine tumor (Fig. 5) were reported as "no abnormality found." In spite of completely uninformative examinations the pain and vomiting were symptoms of intermittent obstruction of the small intestine and operation was indicated.

A right, mid-rectus, muscle-splitting incision was made. The appendix was buried in adhesions and it had a diameter of ¾ inch. It was hard and had a pork-sausage appearance. The terminal 6 inches of the ileum was kinked and fixed in the iliac fossa by a heavy membrane. It was believed that this marked disorder in the lower right abdominal quadrant might not be the cause of her symptoms. Therefore, the entire small intestine was inspected. Three inches distal to the ligament of Treitz, there was a bulbous dilatation of the jejunum due to a movable intraluminal mass. The overlying wall was somewhat edematous and the supplying mesentery was thickened, containing especially prominent veins and lymphatics. Longitudinal enterotomy was performed to disclose an ovoid, semielastic pedunculated lipoma which measured 2¼ by 1⅝ inches in diameter. Its pedicle, which was 2 inches long, was composed of mucosa pulled down from the entire circumference of the bowel. The excess mucosa was excised in an open tubular fashion. The intestinal lining was approximated with interrupted fine chromic sutures; and the enterotomy was closed in a transverse direction. The patient had an uneventful recovery and left the hospital in seven days. She has been perfectly well since then. She commented during a follow-up visit a year later "that operation sure cured me of my psychoneurosis."

The pathologic report of the principal specimen was as follows: Polypoid structure measured 3.5 by 3.5 by 2.5 cm. The surface was covered by a smooth, pink-tan mucous membrane. Five small ulcerations were present in the mucous membrane near the apex, the largest measuring 6 by 2.5 mm. The base of the polyp had attached normal-appearing mucous membrane 7.5 by 4 cm. in size. Section through the polyp revealed the bulk of it to be composed of soft multilobulated bright yellow fatty tissue, covered by the mucous membrane. Microscopic section revealed the mucous membrane to be of the typical small intestine type with a rich component of goblet cells. There was a small sharply circumscribed ulceration which extended to the base of the mucosa and was covered with a fibrinopurulent exudate. Beneath the mucous membrane a relatively thin muscularis mucosae may or may not have been present and external to this, there was a prominent layer of fairly dense fibrous tissue. In the fibrous tissue directly beneath the ulceration there were an abundance of newly formed small blood vessels and moderate plasma cell infiltration. No muscularis propria could be identified. External to the fibrous tissue there was an abundance of fatty tissue of the adult type through which were numerous thin strands of fibrous tissue in which there were blood vessels. The fatty tissue accounted for the grossly described tumor and appeared to occupy the position of the submucosa. (Fig. 6.)

FIG. 6. Case 11. Section of the pedunculated lipoma, showing the normal jejunal mucosa. A part of the large submucosal lipoma is seen deep to the muscularis mucosae. The adult type of fat cell is present.

There are no early symptoms which are pathognomonic of small intestine tumors; their true significance is rarely recognized. In at least a third of the cases[13] they are, at first, diagnosed as due to gallbladder disease, peptic ulcer, chronic appendicitis, pancreatitis and the like; and some have been mistakingly operated upon for these lesions. In the first part of the duodenum and of the jejunum, tumors have symptoms of ulcer or gallbladder trouble. Food or alkali give little or no relief.[11] Periampullary tumors often have jaundice and may have concurrent pain. The icterus may be intermittent or continuous. The associated pain is relieved by vomiting rather than by food. However, adenomas are quite frequent in the duodenum. Here, they rarely attain size large enough to obstruct, but cause ulcer-like symptoms or hematemesis. Tumors low in the ileum may be mistaken for chronic appendicitis or ureteral colic.

In all clinical small intestine tumors there usually are three general syndromes, namely, obstruction, bleeding or perforation. Benign tumors usually produce no symptoms until they intussuscept or bleed, as was present in Case 11. Malignant tumors may have any or all of these symptoms. A pedunculated tumor, as adenoma or polyp, most frequently causes acute symptoms of varying degrees of obstruction because of obturation or intussusception. A stenosing lesion, as carcinoma, which gradually infiltrates the wall, causes slowly progressive obstruction; so that the correct diagnosis is not suspected until the lesion is far advanced. A small carcinoma in the intestinal wall may grow outward into the mesentery, as in Case 1. Here, the physical signs are less evident and the initial symptoms mild. The external mass may cause only distention, nausea, eructations or a vague sense of discomfort. It may compress the intestine late, so that obstructive symptoms are not manifested until there are distant metastases. Benign or malignant growths cannot be differentiated solely by symptoms. Their long duration might indicate benignity of the lesion.

Obstruction of the small bowel is the most common picture seen and it directs suspicion to the correct diagnosis. This was manifested by intermittent cramping and abdominal pain, relieved by vomiting. With jejunal lesions seventy per cent of the patients have some degree of obstruction, duration averaging nine months. In ileal cases 79 per cent have partial obstruction, duration varying from one month to four years, with average duration of 6.3 months.[14] Pain is the most frequent complaint, being present in one-half to two-thirds of the cases. It may be of all types or intensities: cramping or lancinating, sharp or dull; varying from acute to chronic, mild to severe, inter-

mittent to continuous. It usually is located in the epigastrium or around the umbilicus. Pain radiating to the back may be due to involvement of the root of the mesentery. The pain is due to obstruction, either by intussusception or local encroachment upon the lumen of the bowel. More than a third of the patients with benign tumors of the small bowel have symptoms of obstruction.[13] Lipomas and fibromas comprise the highest incidence of these complaints.[13,19]

Vomiting, usually with nausea, is often present with the pain. It is found in up to two-thirds of the cases. Emesis is common in high obstruction. Practically all the food may be emitted soon after eating and this usually relieves the pain. In low obstruction the emesis occurs later and it does not necessarily relieve the distress. Weight loss may be present in benign tumors because of vomiting or decreased intake of food because of the fear of pain. It is more common in malignancies. This loss was present in the second case, which was benign, and not early in the first case, which was malignant.

Bleeding is common in small intestine tumors. It may be low-grade and recognized only on tests for occult blood, or severe enough for transfusions. Melena and anemia are present more often in malignant than in benign tumors. Bleeding is rare as a symptom in carcinoids,[3] lipomas, fibromas and pancreatic rests.[13] It is a common finding in leiomyoma and hemangioma of the small intestine. Hematemesis is a usual symptom in duodenal adenoma. Sometimes the first indication of trouble is a gush of blood or its appearance in the stool, either red, altered or black. Clinically, peptic ulcer and tumors of the colon are the most common sources of intestinal bleeding. When detailed studies of the upper and lower areas of the alimentary tract fail to reveal the origin of the blood, the small intestine should be routinely and carefully examined. Several stool specimens should be tested for occult blood, for the bleeding may be intermittent. Sometimes, examination of the tumor does not reveal any ulceration to account for the melena.[19]

Perforation is not frequent. There usually is a history of preceding vague, abdominal complaints. Adhesions to surrounding loops and localized abscess formation is the rule rather than general peritonitis. The symptoms that result when perforation involves parietal structures mask the indefinite symptoms of a slow intestinal penetration, so that the correct nature of the lesion is often not suspected before operation. Pain in perforation may radiate to the shoulders, back, front of the abdomen or to the testicles.

The physical findings are quite varied, depending upon the size of the tumor and the degree of obstruction. Tumors under the liver shelf are not palpable. A mass may be felt in about 25[15] to 40 per cent[14] of the cases; sometimes, it is the only sign present.[19] Some are palpated only by pelvic examination.[20] The tumor may seem to slip away from the fingers because palpation may push it among the loops of intestine. The location of jejunal masses is predominantly in the lower left quadrant; while of those in the ileum, more than two-thirds are in the lower right quadrant. Sarcoma may be palpated more readily than carcinoma. This is explained by their usual larger size.[8] They grow outward into the mesentery, so that they more often attain largeness before the patient is aware of trouble. With clinical obstruction present, visible peristalses are seen in one-third to one-fourth of the cases. Borborygmi are heard in about half of all cases.

Roentgen examination offers a high ratio of preoperative diagnosis of tumor, yet the growth may be overlooked in a few.[19] Up to 90 per cent of lesions of the small bowel may be suspected upon the basis of adequate x-ray examination.[14,21] Although lesions other than tumor may simulate some of the changes, filling defects due to tumor are generally sharply demarcated at both the proximal and distal margins and involve a short segment. There is alteration or obliteration of the mucosal pattern at the site of the lesion; and there may be obstruction and ulceration.[17] Tumors in the submucosa expand the mucosa to iron out or obliterate the mucosal pattern. In the early stage of obstruction, such as that produced by a constricting neoplasm, the dilatation may not be pronounced and can be demonstrated for only a short distance proximal to the tumor. The characteristic appearance of obstruction with severe dilatation is usually absent unless there be sudden onset such as that produced by intussusception of a polypoid tumor. This is due chiefly to the fact that the small intestine has the inherent ability to compensate for a gradual reduction in

diameter of the lumen.[17] One may see only a dilated segment just above an area of constriction, or there may be only a dilated portion with gas or barium without actually recognizing any outline of a lesion.

There is no reliable roentgenologic evidence of benignancy once the evidence of tumor is produced. There may be all the accepted morphologic evidence of benign tumor, as definitely circumscribed, regular in contour, yet microscopic examination of the lesion may show it to be malignant.[21] In lymphosarcoma of the small intestine, positive evidence of a tumor causing obstruction was seen in 64 per cent of the cases.[6] The small intestine enema gives a filling of the whole small intestine to disclose filling defects. A small duodenal tube may be used to control the instillation of the barium suspension better. While serial plates require much time, special care may be well rewarded by revealing a tumor which might have been missed by the usual studies in the gastrointestinal series. Lesions of the lower ileum may be revealed by reflux through the ileocecal valve during barium enema. In leiomyoma there often is a central niche due to necrosis.

The most common tumor of the small bowel is carcinoma,[16,20,21] followed in incidence by benign tumors and then carcinoids. Carcinoma of the whole colon is forty times more common, and carcinoma of the stomach is about one hundred times more frequent, than that of the small intestine. The latter is twice as common in males as in females. It occurs mostly between the third and seventh decades. The larger number of patients are in the fifth and sixth decades than in any other ten year span.[14] Authors differ on the most common site of carcinoma; some say the ileum,[8] the jejunum,[10,12] the duodenum,[11] and, in the last area, the ampulla of Vater is about the only part involved.[2,16]

The malignancies of the small intestine are carcinoma and other neoplasms, such as carcinoids, malignant myomas and sarcoma.[14] The types of carcinoma are the adenomatous, which occurs in about 90 per cent of the operated cases, colloid and undifferentiated cell carcinoma. Rarer lesions may be melanocarcinoma and scirrhous carcinoma. Sarcomas are neurogenic sarcoma, leiomyosarcoma and lymphosarcoma. The adenocarcinoma usually has a gland-like structure closely resembling the mucous glands of the small intestine. The colloid carcinoma may have abundant extracellular mucoid or colloid material which distends the lumina of the glandular structures, compressing the tumor cells at the periphery. There may be greatly distended cells forming signet ring cells with nuclei somewhat crescent shaped, eccentrically placed and deeply stained. Metastasis occurs early, first in the mesenteric nodes and peritoneum, then liver, lungs, long bones, etc. The infiltrating or undifferentiated cell carcinoma has tall columnar epithelium, sometimes polygonal, arranged in nests and strands packed together. There is no definite pattern formation.

There is obstruction of varying degree, with dilatation and hypertrophy above the lesion and relative constriction below it. Nodal involvement is related to the grade of malignancy. There averaged 20 per cent nodal involvement in grade I lesions to 81 per cent in grade IV. The larger the lesion, the greater is the incidence of nodal involvement and the higher the grade of the lesion.[14] The enlarged masses may extend to the root of the mesentery so that complete extirpation is impossible. Malignancies of the small intestine are usually single. There are quite rare occurrences of two coexistant malignancies, where the second growth is either in the small intestine or in the stomach or colon.[7] Sarcoma and carcinoids may be multiple and are found more frequently in the ileum.[20] In localized gastrointestinal lymphosarcoma, or even with metastasis, blood studies do not reveal a leukemic picture.

Carcinoids are neoplastic growths of the argentaffin cells which lie in the crypts of Lieberkühn, deep in the mucosa of the gastrointestinal tract. About 90 per cent of all carcinoids of the intestine originate in the appendix or terminal ileum, most of them being in the appendix. About 14–23 per cent of all malignancies of the small intestine are carcinoids.[3] In the appendix they are about four times more common in the female; while the extra-appendiceal ones are about equally divided between the sexes. The average age for the first is about the third decade; while, in the second type, the average is highest around the sixth decade.

Carcinoids have a dual personality, depending upon their site. Those in the appendix rarely metastasize; while the extra-appendiceal

ones metastasize in up to 40 per cent of cases. Benign argentaffinomas are usually submucosal, freely movable, and are usually smaller than 1 cm. They are grossly encapsulated, yellowish in color and rarely ulcerate. Malignant ones may be small and tend to invade muscle and serosa, causing puckering. The adjacent spread in the mesentery usually is large so that it is sometimes palpable. The distant metastases are to regional lymph nodes and to the liver.

When carcinoids are localized, there is no clinical evidence of trouble in 90 per cent of cases.[3] In the malignant form there are obstructive symptoms in 50 per cent. These are very slow growing and some patients have symptoms for over a decade.

The benign tumor group is composed of adenoma, which is the most frequent, followed by fibroma or leiomyoma, lipoma, neurofibroma, cysts, hemangioma, pancreatic rests, etc. They occur in all parts of the small intestine, being more common in the ileum, according to many reports, and in the jejunum in others. A fourth of all the benign tumors of the intestinal tract are found in the small intestine.[16] The majority of adenomas are polypoid and benign. It is estimated that 7 per cent of these become malignant.[11] Adenomas of intestinal or of Brunner's glands occur in the duodenum.[1]

Of the lipomas in the gastrointestinal tract, more than one-half are found in the small intestine. They are most common in the ileum,[13,19] and most rare in the stomach. They are evenly divided between the sexes.[18] The majority occur between the third to sixth decade, so that seventy-five per cent are found during the so-called cancer age. Lipomas are either submucous or subserous in position, and both types may coexist. In about 90 per cent they are submucosal and they usually occur as single tumors.[18] As other benign tumors they may be multiple or diffusely lipomatous, sessile or polypoid. A high percentage of sizable small intestine lipomas cause intussusception.[18,19] These vary from 2 to 10 cm. in diameter. Asymptomatic lipomas, usually measured in millimeters, are found unexpectedly during operation for other unrelated conditions and during autopsy.[5,13,19]

The fat cells are all adult type, although some immature fat cells may be in the periphery of small lipomas.[5] Even small tumors have septa dividing them into lobules. The overlying mucosa may be normal, thinned, atrophic or ulcerated. In small growths the muscularis mucosae may be traced intact over the tumor. In large tumors, it may not be recognizable. The muscular layer is not affected by small tumors. In large growths this layer may be so thinned that the yellow color is apparent externally. The tumor mass may become edematous, congested, ulcerated, or necrotic. There are rare instances of spontaneous extrusion of lipomas through the anus.

The treatment is determined by the type of lesion and the condition of the patient. Any tumor found which projects into the lumen of the bowel should be removed because of the threat of intussusception. When identity of the tumor is not known, benign or malignant, it is better to resect the segment. In pedunculated benign tumors enterotomy and ligation of the pedicle will suffice. Other benign tumors are treated by resection of the involved segment. When a reducible intussusception is found, one may resect only the tumor. Or, if the intestine is edematous, either with a reducible or unreducible mass, the segment involved may be excised and intestinal continuity restored if the patient's condition permits. If the condition of the patient is poor, so that resection should not be performed, the lesion may be exteriorized; and secondary operation performed later. There is the danger of a leak if the anastomosis is performed with inflamed or edematous bowel.

In exploration for cause of hematemesis and melena, when external examination of the intestine fails to reveal a tumor, it may be necessary to open and palpate the duodenal mucosa. A benign adenoma is a common cause of duodenal bleeding.[1] This growth is so soft that it can be identified only by digital examination within the lumen. In the absence of intestinal obstruction hematemesis indicates bleeding proximal to the ligament of Treitz.

Carcinoma should be resected with a long margin of clear bowel, if possible, removing a wedge sector of its mesentery. Intestinal continuity may be re-established by end-to-end or side-to-side anastomosis. When there is massive involvement of the mesentery, preventing excision, a symptom-relieving procedure as a sidetracking operation should be done rather than to close the abdomen helplessly. In high lesions one can do a palliative gastro-

enterostomy or enteroenterostomy around the lesion. The results are better in resecting carcinoids than in other malignancies. Drastic surgery may get a cure if all the visible neoplasm can be excised. This tumor is slow growing, so that palliative anastomosis around extensive involvement of the intestine has resulted in unexpected long clinical relief.

Some believe all sarcoma cases, whether operable or not, should have x-ray therapy. Although it has no, if any, effect on duration of life, it alleviates symptoms, diminishes size of the tumor and improves feeling of well-being.[8] Irradiation therapy serves to eradicate small foci of tumor tissue remaining after operation for lymphosarcoma.[6]

Resection can be done in about 45 per cent of cases of tumor of the small intestine. The surgical mortality in lymphosarcoma ranges from 12 per cent[6] to 30 per cent,[12] and in sarcoma to 60 per cent.[8] The rate is highest in the duodenum and lowest in the jejunum.[12] Average duration of life after successful operation for malignancy ranges from seventeen months[20] to about three years[14] or longer. While cancer of the colon has 50 per cent five-year survivals,[4] in the small intestine it is 12 per cent.[12]

SUMMARY

Two cases are reported of tumors of the small intestine, adenocarcinoma and pedunculated submucous lipoma, both occurring close to the ligament of Treitz. The initial difficulties in diagnosis are described.

A review is presented of the symptoms, roentgenograms, pathology and surgical treatment of tumors of the small intestine.

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Present Day Surgical Management of Acute Disease of the Gallbladder

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TWENTY years ago in a discussion of the treatment of acute cholecystitis Finney[9] told of the early development of biliary surgery. "The gallbladder was exposed and sutured to the parietal peritoneum. Then, after waiting for a few days, a small opening was made in the organ and it was let drain, without attempting to remove the gallstones. Then some bold soul began removing the gallstones, and so cholecystostomy was established."

For many years cholecystostomy was the treatment of choice for all cases of acute cholecystitis. In 1923 Walton[15] of England advocated early operation for acute cholecystitis. Consequently many papers were published recommending early or delayed operation. At the height of the controversy in 1933 mortality figures in operations for acute cholecystitis varied from 9.3 per cent reported by Smith,[14] who advocated delayed operation, to 5.2 per cent published by Judd and Phillips,[11] who advocated early operation.

Considerable confusion arose over what was meant by "early operation" and this confusion existed up to recent years. Marshall[13] in a discussion of early and late operation speaks of "early" as "within 72 hours to the complete cessation of clinical manifestations," and "late" as "anytime after clinical remission of objective and subjective symptoms."

Cole[6] believes immediate operation can be performed if the patient is seen within thirty-six to forty-eight hours after the onset of symptoms. On the other hand Buxton and associates[3] state that the condition of the patient at the initial examination is of more importance than the duration of the acute disease.

A series of seventy-seven consecutive private and ward patients treated surgically for acute disease of the gallbladder here recorded represents 6.4 per cent of 469 operated patients with diseases of the biliary tract.

INCIDENCE OF ACUTE CHOLECYSTITIS

The wide variation in the incidence of acute disease of the gallbladder as reported throughout this country by Adams and Stranahan[1] (5 per cent), Buxton and associates (1.5 per cent) and Heuer[10] (23.5 per cent) is not readily explained. However, in 1935 Andrews[2] called attention to the confusion existing in the pathologic classification of diseased gallbladders. Many of the cases are an acute exacerbation of a chronic cholecystitis and, according to Andrews, even in acute cholecystitis the predominating factors are edema, hemorrhage and infiltration with large and small mononuclear cells and very few polymorphonuclear leukocytes. It is, therefore, easily understood why the surgeon's diagnosis at operation should frequently differ from the pathologist's report. This difference in diagnosis could be reflected in statistical studies.

DIAGNOSIS OF ACUTE CHOLECYSTITIS

Of the seventy-seven cases, forty-eight (62.3 per cent) patients had a history suggestive of gallbladder disease and only four gave a history of jaundice. Tenderness to palpation, either in the epigastrium or right upper abdominal quadrant was present in seventy-three patients (94.8 per cent), while four had generalized abdominal tenderness. Abdominal rigidity was present in twenty-four (31.2 per cent) and a mass in the right upper abdominal quadrant could be felt in twenty (25.9 per cent). There was an elevation of temperature present in fifty-two (67.5 per cent) of the cases and an elevation of the leukocyte count in forty-seven (61 per cent).

In the presence of jaundice and hepatitis a cholecystogram is contraindicated. If the patient is acutely ill and intestinal suction must be carried out, a cholecystogram cannot be done. A scout film of the abdomen is indicated especially if a perforated ulcer or kidney stone is suspected. Table 1 lists the roentgen exami-

nations made in this series. All of the nine scout films were reported negative for gallstones. It is well to remember that a normal cholecystogram may occur even in the presence of acute cholecystitis. Therefore, the clinical impression is more important than the roentgenogram.

TABLE I
ACUTE CHOLECYSTITIS
PREOPERATIVE ROENTGEN EXAMINATIONS

Scout films of the abdomen	9
Non-functioning gallbladder	20
Non-functioning gallbladder with calculi	4
Biliary calculi	5
Subnormal response to dye	3
Subnormal response with calculi	6
Normal cholecystogram	1
Total	48

PERFORATION OF THE GALLBLADDER

Perforation of the acutely diseased gallbladder has long been considered a major complication. In fact, in the older literature the percentage of perforation was high. Judd and Phillips[12] in 1933 reported sixty-one perforations in 508 cases (12 per cent) of acute cholecystitis. There was abscess formation about the wall of the gallbladder in thirty-eight cases. However, there were only three cases of free perforations into the peritoneal cavity with subsequent peritonitis. In a more recent series Clagett[5] reported 13 per cent perforations, Heuer 20 per cent and Cowley and Harkins[4] 4.7 per cent. We found eight cases, 10.3 per cent, of perforation of the wall of the gallbladder. Of these, three were free perforations into the peritoneal cavity. One patient was operated upon the day of admission, one on the second hospital day and the other on the fourth day. In none of these patients did generalized peritonitis develop. Of the adherent perforations, in one patient a cholecystogastric fistula had developed.

SURGICAL INTERVENTION

After a presumptive diagnosis is established efforts are directed toward the hydration of the patient with intravenous fluids. An hourly chart determines any sudden change in temperature or pulse. If the history is of less than forty-eight hours' duration, immediate operation when the patient's condition permits is planned. Antibiotics are administered as soon as the patient is admitted to the hospital. If the disease seems to be subsiding or stationary, as evidenced by a stable temperature and pulse rate, and localized abdominal tenderness, the operation may be postponed until an icterus index and prothrombin time have been determined and roentgenographic examinations are made. No patient is operated upon if the hemoglobin is below 10 gm. Anesthesia is not administered before a Levin tube is placed in the stomach and nasogastric suction instituted. The fact that forty-nine patients (63.6 per cent) in this series gave a history of more than forty-eight hours' duration indicates the problem that confronts the surgeon. Operation was performed on the day of admission in eleven patients (14.3 per cent) and within forty-eight hours in twenty-four (31 per cent).

TABLE II
OPERATIONS FOR ACUTE CHOLECYSTITIS*

	No. of Cases	Per cent
Cholecystectomy	61	79.2
Cholecystectomy with choledochostomy	4	6.7
Cholecystostomy†	12	15.6
	77	100

* No deaths in this series. Four hundred sixty-nine cases of diseases of the biliary tract; 14.7 per cent choledochostomy.

† Cholecystostomy cases returned for cholecystectomy: with duct exploration, three; without duct exploration, two.

All the patients in this series were operated upon. (Table II.) Of the sixty-five cholecystectomies, fifty-seven were performed in a retrograde manner, and eight, because of technical difficulties, were done by freeing the fundus of the gallbladder from above in order to facilitate identification of the cystic duct and artery. This latter procedure should be used without any hesitation, especially in markedly obese patients when exposure is difficult and when the gallbladder is edematous. A line of cleavage is readily obtained by incising the edematous serosal layer of the gallbladder. The organ can then be rapidly removed from its bed by blunt dissection. Some bleeding may be encountered from the gallbladder bed. This can be controlled with hot packs and by suture. Hemostatic substances to control hemorrhage in the gallbladder bed

should be used with caution. In two instances in which we employed gelfoam subphrenic abscess developed.

In acute cholecystitis exploration of the common duct can be done, when indicated, provided the patient is a good risk. However, cholecystostomy should be done in the poor risk patient. Buxton and co-workers rightly stress the value of this procedure, especially if identification of the common duct is difficult. It is also indicated in poor risk patients and in the aged. Since 1945 we have used this procedure in twelve patients. We believe that with the advent of antibiotics and with improvements in preoperative preparation and anesthesia the indications for cholecystostomy are fewer. Nine of the twelve cholecystostomies were done before 1948. Of the twelve cases, five (41 per cent) returned for cholecystectomy. Of the remaining patients, six have been followed for from one to five years without symptoms.

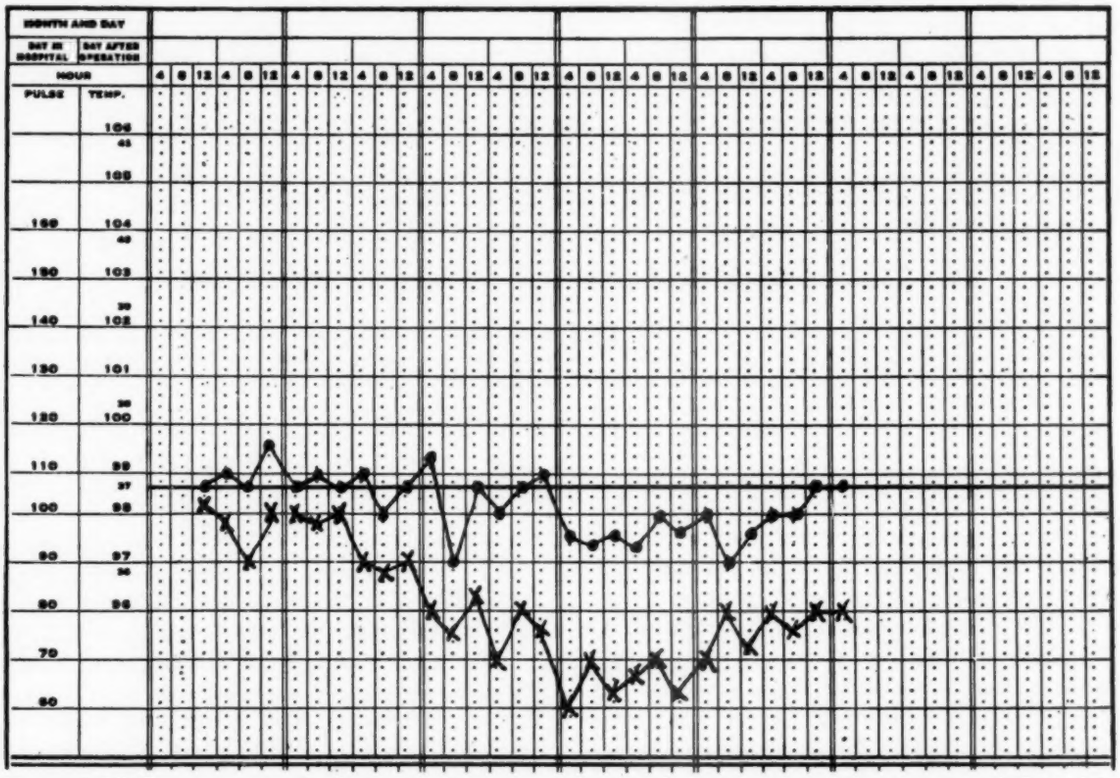

FIG. 1. J. McC., age twenty-six. Postoperative course following cholecystectomy for gangrenous cholecystitis.

MORBIDITY

A study of the records in this series was made to determine whether the advent of antibiotics had changed the postoperative course. Penicillin therapy was instituted in 1946, and since then has been given routinely to all patients with acute cholecystitis. Streptomycin has been used since 1948. Since more than 50 per cent of sterile cultures are obtained from acute disease of the gallbladder, antibiotic therapy is necessarily largely empirical. We are impressed with the value of the newer antibiotics with a larger spectrum. Since 1950 the average postoperative temperature curve has been normal on the third day as compared with the seventh day in 1945. Early ambulation has been an aid in reducing the number of hospital days.

The fact that many of these patients, especially when operated upon early in the course of the disease, have a comparatively uneventful postoperative course, is evidenced by the following case history.

CASE REPORT

J. McC., aged twenty-six, was admitted to the Lankenau Hospital complaining of epigastric pain and vomiting of twenty-four hours' duration. The pain radiated along the right costal margin to the back. There was no previous history of pain, indigestion or intolerance to fatty foods. The patient appeared

acutely ill. The temperature was 99°F., pulse 110, respirations 20 and blood pressure 120/80. Abdominal examination revealed board-like rigidity of the right upper quadrant extending to the midline. Peristalsis could not be heard. The red blood cell count was 4,600,000, the hemoglobin 14.6 gm. and the white cell count was 19,750 with 76 per cent polymorphonuclear leukocytes. A scout film of the abdomen was normal. The resident surgeon's impression was "perforated peptic ulcer." The patient was prepared for operation with adequate intravenous fluids. Operation revealed a gangrenous gallbladder with an impacted stone in the cystic duct. The patient received two million units of penicillin and 7 gm. of streptomycin postoperatively.

Evidence of the uneventful recovery of this patient is shown in Figure 1.

COMMENTS

Although early operation is still preferable in the treatment of acute cholecystitis, the majority of patients with this condition continue to come to the hospital more than forty-eight hours after the onset of the disease, 61 per cent in this series. Accordingly, until these patients are seen earlier, it will continue to be necessary to individualize in the surgical treatment of acute cholecystitis. Of paramount importance is the condition of the patient and not the length of time that the disease has progressed. The frequent comparatively afebrile postoperative course after cholecystectomy for acute cholecystitis substantiates the studies published by Feinblatt[8] and Andrews which showed that the underlying lesions in cholecystitis are vascular, due to obstruction from impaction of a stone in the ampulla. Denton[7] also called attention to the fact that the lesions in acute cholecystitis are like a "hemorrhagic infarct," with edema, hemorrhage and infiltration with large and small mononuclear cells. Accordingly, infection is probably secondary. The greatest hazards in surgery of acute disease of the gallbladder are those resulting from edema and adhesions around the common bile duct. This will continue to be a pitfall for the inexperienced or hasty surgeon.

SUMMARY

An analysis of seventy-seven consecutive patients with acute cholecystitis, representing 6.4 per cent of 469 patients operated upon for diseases of the biliary tract, treated by cholecystectomy and cholecystostomy is presented.

There were no operative or postoperative deaths.

Early and delayed operation, cholecystostomy and perforation of the gallbladder are discussed. Problems arising over the diagnosis of acute cholecystitis are presented.

CONCLUSIONS

1. Cholecystectomy for acute cholecystitis, whether the operation is early or delayed, has a low operative mortality at the present time.
2. Early operation is still preferable, but there is a trend toward individualization of each case.
3. Perforation of the gallbladder in acute cholecystitis does not play a major role in the present day management of acute cholecystitis.
4. Cholecystostomy should be done for poor risk and aged patients although indications are fewer than in former years.

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The Overuse of Carbon Dioxide Absorption in General Anesthesia

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THE publication in 1915 by Dennis E. Jackson of a description of a method of absorbing carbon dioxide from the exhalations of any human subject, and its perfection and adaptation to gas machines commonly used in the administration of gas-oxygen anesthesia by Waters in 1924, marked one of the outstanding advances in the art of anesthesia made during the first half of the present century. In 1928 Guedel invented the inflatable cuff for the administration of air-tight closed endotracheal administration of gaseous anesthetic agents.

The invention and adaptation to clinical needs of the carbon dioxide absorption technic was notable, not because it enabled the anesthetist to remove all of the "toxic" carbon dioxide from the anesthetic gases but because by removing carbon dioxide hyperpnea was prevented. Hitherto the latter had been the "bugbear" of smooth gas-oxygen anesthesia.

Unfortunately for the vast majority of patients currently undergoing major surgical intervention, the pendulum has swung much too far in the direction of too complete and too efficient removal of carbon dioxide from the respiratory anesthetic gases. This militates against the immediate and postoperative welfare of a surgical patient. The exception to this generalization is Guedel's controlled respiration technic invaluable in thoracic surgery.

PHYSIOLOGIC CONSIDERATIONS

The exponents of the complete removal of carbon dioxide school of general anesthesia (and admittedly they are the vast majority of present day teachers of anesthesia, notable exceptions being Guedel and Lundy) take the view that carbon dioxide is a poisonous gas that should be immediately and completely removed from the gases respired by a surgical patient. They have no difficulty in accomplishing this due to the efficacy of soda lime as an absorber of carbon dioxide. The writer is in diametric opposition with this view of the alleged toxicity of even small quantities of carbon dioxide. On the contrary, I believe that the complete removal of carbon dioxide from the anesthetic gases breathed by the surgical patient is definitely prejudical to the best interests of that patient. This opinion, although extremely unorthodox among anesthesiologists at this time, will be supported by the following physiolgic and clinical data. (Tables I and II.)

TABLE I[1]

OXYGEN AND CARBON DIOXIDE TENSIONS IN ALVEOLAR AIR AND BLOOD

	Oxygen Tension (mm. Hg)	Carbon Dioxide Tension (mm. Hg)
Alveolar air	100	40
Arterial blood	80–90	40
Mixed venous blood at rest	40	46

TABLE II[1]

COMPOSITION OF ALVEOLAR AIR

Carbon dioxide	4.7 to 6.4% (average 5.6%)
Oxygen	14.0% (approx.)
Nitrogen	80.0%

In daily life our breathing is constantly and automatically adjusted to current needs by the action of carbon dioxide upon the respiratory center to such effect that: (1) we are assured of a respiratory intake of 250 cc. of oxygen per minute and (2) we exhale or, if our physiologic needs demand it, retain quantities of carbon dioxide suitably gauged to the purpose of maintaining the H+ ion concentration of the blood at approximate neutrality, i.e., at a pH of about 7.35.

When for any reason there is a tendency toward acidemia, carbon dioxide is profusely exhaled in order to rid the blood of acid ions of carbonic acid. Contrariwise, when there is a tendency toward alkalemia, carbon dioxide is

retained in the blood in order to counteract the alkalemia.

The terms acidemia and alkalemia have more meaning to us as clinicians if, with Rowntree, we recall that "when our blood becomes as acid as triple distilled water we will likely die *in coma;* when it becomes as alkaline as ordinary tap water we are likely to die *in convulsions.*"

When hyperpnea is induced by any means, an excessive loss of carbon dioxide occurs. This loss of the acid-forming carbon dioxide, if severe enough, will lead to alkalemia and thus to convulsions. As will be discussed hereafter, a direct relationship exists between cerebral hypoxia, alkalemia and convulsions.

Obviously, when gas-oxygen anesthesia is administered with the carbon dioxide absorber on fully, as is usually the case, the normal regulatory action of carbon dioxide upon respirations and blood reaction is completely frustrated.

Gaseous Exchanges in the Pulmonary Alveoli. When one compares the respective tensions of both oxygen and carbon dioxide in the alveolar air and the arterial blood (Table I), it is apparent that the pressure differences are such that gaseous exchanges in the lungs are promoted by a simple process of diffusion through the pulmonary epithelium. For instance, the oxygen tension of alveolar air is about 100 mm. Hg while that of venous blood is only about 40 mm. Hg, leaving a pressure differential of 60 mm. Hg on either side of the alveolar membrane.

In respect to carbon dioxide the corresponding tensions on either side of the alveolar membrane are 40 and 46 mm. Hg, respectively, the pressure difference between venous blood and alveolar air being only 6 mm. Hg. However, carbon dioxide passes through the pulmonary epithelium with much greater ease than does oxygen to such effect that even a small difference of 6 mm. Hg in gaseous tensions is sufficient to enable the lungs to eliminate about 10 L. of carbon dioxide per minute if necessary. On the other hand, oxygen passes through the pulmonary epithelium with relative difficulty, the oxygen tension of arterial blood being 10 to 20 mm. Hg lower than that of alveolar air.

Regulation of the Rate and Volume of Respiration. The regulation of the rate and volume, or depth, of respiration is largely controlled by the action of carbon dioxide in a quantitative manner upon the respiratory center. (Table III.)

A study of the data contained in Table III suggests that: (1) At sea level and breathing fresh air the normal respiratory rate is 14 per minute and the average volume is 673 cc.; under these circumstances the percentage of carbon dioxide in the alveolar air, and therefore

TABLE III[1]
(AFTER HALDANE AND PRIESTLY)

Percentage of Carbon Dioxide in Inspired Air	Average Depth of Respiration (cc.)	Average Frequency per Minute	Ventilation of Alveoli in cc. of Inspired Air (cc.)	Percentage of Carbon Dioxide in Alveolar Air
0.04	673	14	100 (as normal)	5.6
0.79	739	14	116	5.5
2.02	864	15	153	5.6
3.07	1216	15	226	5.5
5.14	1771	19	498	6.2
6.02	2104	27	857	6.6

in the arterial blood, is constantly about 5.5 to 5.6 per cent. (2) When the carbon dioxide content of the inhaled air, or the anesthetic gases, is increased seventy-seven fold from 0.04 to 3.07 per cent, the respiratory rate *per se* increases only by one per minute while the respiratory volume is almost doubled. (3) It is only when the carbon dioxide content of the inspired air or anesthetic gases reaches 5.14 per cent and the rate of breathing rises to 19 per minute that any appreciable increase occurs in the carbon dioxide content of the arterial blood or alveolar air; that increase is only 0.7 per cent.

Studied consideration of these physiologic data suggests, among other things, that as anesthesiologists we may well permit carbon dioxide to accumulate in the rebreathing bags of our gas machines to about seventy-seven times the amount currently permitted with no harm and much benefit (i.e., doubling of the respiratory volume) accruing to the surgical patients under our care.

Although many of the leaders in anesthesiology in the United States, Britain and Canada are presently of the opinion that even small excesses of carbon dioxide are toxic, it is a matter of record that Haldane[2] has shown experimentally that concentrations of carbon dioxide as high as 6.2 per cent can be rebreathed indefinitely—provided adequate oxygen is supplied—with no more serious effect upon the human subject than a severe headache.

Oxygen Transport and Oxygen Dissociation. The hyperbolic curve for oxyhemoglobin re-

duction shown in Figure 1 indicates graphically how unsuitable hemoglobin would be as a carrier of oxygen if it were to act in the tissues in the manner that it acts in the laboratory, as delineated by this curve. It would show great avidity for oxygen on exposure in the lungs but

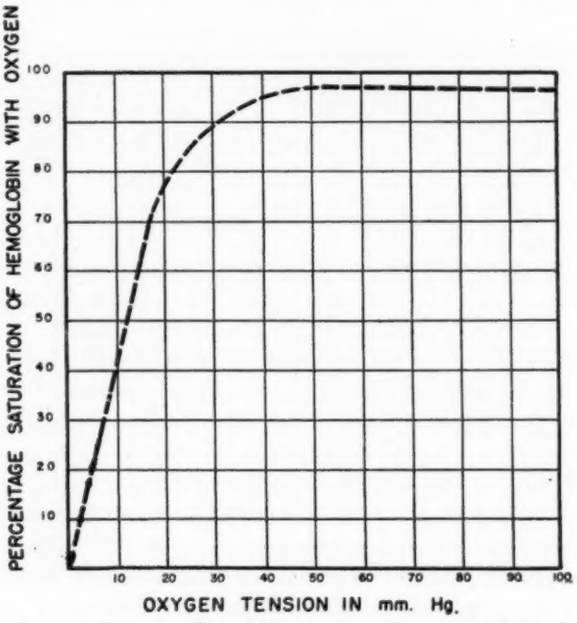

FIG. 1. Oxygen dissociation curve for a solution of hemoglobin. (From: BEST, C. and TAYLOR, N. The Physiological Basis of Medical Practice, 5th ed. Baltimore, 1950. Williams & Wilkins Co.)

it would not yield up its load of oxygen in the tissues until the surrounding partial pressure of oxygen had fallen to a very low level indeed. For this reason, under the partial pressure of oxygen that normally exists in the tissues the process of dissociation of oxygen from hemoglobin would be many times slower than its rate of formation in the lungs, i.e., about $\frac{1}{100}$th of a second. This would render hemoglobin useless as a carrier of oxygen to the tissues.

Thanks to the provisions of Nature, the mere presence of carbon dioxide in the arterial blood and in the tissues enables the dissociation of oxygen from oxyhemoglobin to keep pace with the oxidation of hemoglobin in the pulmonary alveoli. (Fig. 2.) This vitally important role of carbon dioxide in speeding up the dissociation of oxyhemoglobin was first disclosed by Bohr and is accordingly termed "the Bohr effect."[3]

On reference to Figure 2 it will be noted that with the rise of carbon dioxide tension in the tissues the dissociation curve of oxyhemoglobin flattens out toward the right. This indicates that in the presence of carbon dioxide the affinity of hemoglobin for oxygen is released and oxygen thereupon becomes available for tissue respiration. Guedel[4] has estimated

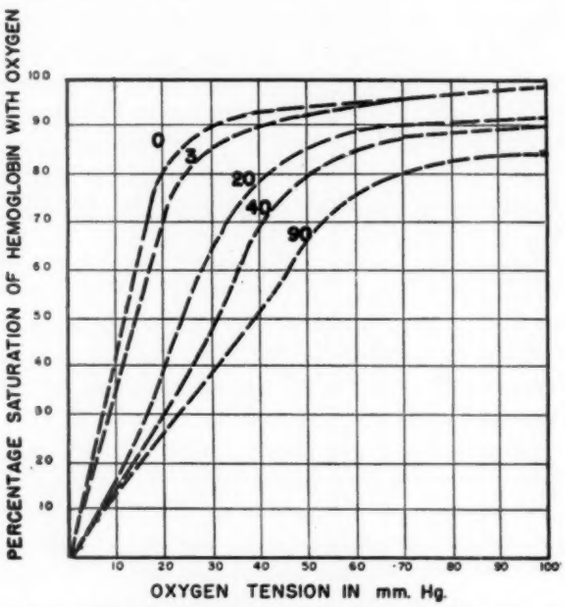

FIG. 2. Dissociation of human blood exposed to 0, 3, 20, 40 and 90 mm. Hg tensions of carbon dioxide. Ordinates: percentage saturation. Abscissae: oxygen pressure. (After Barcroft, from BEST, C. and TAYLOR, N. The Physiological Basis of Medical Practice, 5th ed. Baltimore, 1950. Williams & Wilkins Co.)

roughly that when the carbon dioxide tension in the arterial blood reaches 90 mm. Hg, an extra 25 or 35 cc. of oxygen per minute are made available for tissue respiration.

In summary, we might well agree with Meischner[1] who said, "Over the oxygen supplies of the body carbon dioxide spreads its protective wings."

CLINICAL DATA

Convulsions Related to Hypoxia and Alkalemia. The direct relationship that exists between hypoxia and alkalemia on the one hand and convulsions on the other is well known to most clinicians but apparently has escaped the notice of many anesthesiologists, particularly those who advocate complete carbon dioxide absorption in gaseous anesthesia. "Deep ether convulsions," sometimes termed "late ether convulsions," which are occasionally seen under open or semi-open ether anesthesia in

the third stage, illustrate this point. The following case history is typical of eight others reported by Kemp[5,6] between 1932 and 1944:

This patient was a thirty-five year old man seen in 1931. Laparotomy and primary closure of a perforated gastric ulcer were performed. Anesthesia was administered through endotracheal insufflation of ether vapor with oxygen and without rebreathing. Anesthesia had continued uneventfully for thirty-five minutes and the surgeon was suturing the peritoneum when convulsive muscular movements began in the right side of the face, in the right upper extremity and in the abdominal muscles of the right side. The administration of ether vapour was immediately discontinued and the mask of a gas machine, with attached rebreathing bag, was applied to the patient's face thus forcing him to rebreathe an atmosphere of oxygen to which the carbon dioxide of his own exhalations was gradually added. (There was no other available source of carbon dioxide.) Within five minutes the convulsive movements ceased entirely and the abdominal closure was completed. A catheter specimen of urine taken at that time was found to be "definitely alkaline in reaction." The patient made an uneventful recovery from the operation and anesthesia.

When reporting the aforementioned and seven similar cases, Kemp[5,6] ascribed the convulsions to cerebral hypoxia brought on by excessive loss of carbon dioxide due to hyperpnea in the cases under semi-open or semi-closed ether administration or, as in the case just cited, from acapnia due to loss of carbon dioxide induced by the open type of endotracheal anesthesia being used. In other words, the convulsions described were undoubtedly due to an absence of the salutary Bohr effect of carbon dioxide in furthering the quick release of oxygen from oxyhemoglobin in the cerebral tissues.

However, it is only fair to point out that the majority of anesthesiologists reporting "deep ether convulsions" or describing them in textbooks take different views. Some of them even go so far as to blame excess carbon dioxide for the convulsive movements. That this latter concept is in direct antithesis to modern physiologic teaching is obvious.

In the field of neurosurgical research Penfield and Erickson[7] have shown that basically all convulsions are due to cerebral hypoxia. In many cases of epilepsy convulsions can be readily induced by overventilation while the patient is in the operating room under block anesthesia. These authors have described typical grand mal attacks induced by pulmonary overventilation and preceded by a blanching of the cerebrum at the point of "focus" of the epileptic seizure.

In explanation of these clinical phenomena Penfield and Erickson ascribe the onset of the grand mal attack to hypoxia of the cerebral tissues brought about by alkalemia induced by excessive loss of carbon dioxide; in other words by the absence of the Bohr effect of carbon dioxide in releasing oxygen from oxyhemoglobin in the tissues of the cerebral cortex. They have stated, "The plasma pH has been observed by a number of investigators to shift towards the alkaline side just prior to the onset of the self-induced seizures."

In support of this opinion of the hypoxic origin of convulsions in humans Penfield and Erickson[7] point out a clinical phenomenon that has been reported by others in connection with cardiac asystole. During cardiac asystole there is a continuance of respiration with the result that the stagnated blood in the lungs becomes overventilated and all its carbon dioxide is washed out. When cardiac function is resumed, this alkaline blood (alkaline because it is very low in carbon dioxide content) circulates through the respiratory center and creates a period of apnea. Cerebral hypoxia, together with low carbon dioxide tension in the cerebral arterial blood, causes convulsions to occur after cardiac function has been restored. If oxygen and 5 per cent carbon dioxide are inhaled with the onset of the asystole, the convulsions do not occur.

Consideration of the foregoing data (convulsions under ether anesthesia, convulsions after hyperpnea in epileptics and convulsions following cardiac asystole) adds clinical confirmation to the physiologic concept that carbon dioxide must be present in the arterial blood in adequate amounts for the dissociation of oxygen from oxyhemoglobin to occur. If it is not present, alkalemia and tissue hypoxia and convulsions are likely to occur.

Convulsions under Gas-Oxygen Anesthesia. Taylor[9] has reported the fact that in 33,777 administrations of cyclopropane and oxygen in the Wisconsin General Hospital and Bellevue Hospital in New York, apnea requiring respiratory stimulation occurred in 248 cases (0.7 per

cent) and convulsive movements were seen in thirty-nine other cases (0.1 per cent).

Kemp[7] has reported two cases of convulsive movements under gas-oxygen anesthesia in which the convulsive movements are ascribed to hypoxia of the cerebral cortex brought on by (1) the low tidal volume of pulmonary ventilation and (2) overenthusiastic withdrawal of all carbon dioxide from the respired anesthetic gases. Is it not possible, or even probable, that a similar explanation would apply to the 248 cases of apnea and the thirty-nine cases of convulsions reported by Taylor?

SUMMARY AND CONCLUSIONS

1. A certain minimal tension of carbon dioxide must be present in the arterial blood for the normal release of oxygen from oxyhemoglobin.

2. It is suggested that convulsions seen under deep ether anesthesia, convulsions seen in epileptics after self-induced hyperpnea and convulsions seen in patients recovering from cardiac asystole are all due to the same basic cause: hypoxia of the cerebral cortex induced by excessive loss or removal of carbon dioxide from the arterial blood.

3. Clinical reports and clinical experience in anesthesia suggests that the too efficient withdrawal of carbon dioxide from the anesthetic gases breathed by a patient undergoing major surgical intervention is definitely not in the best interests of that patient.

4. Quantities of carbon dioxide should be allowed to accumulate in the rebreathing bags of gas machines to approximately 3 per cent of the respired gases in the interests of maintaining a normal Bohr effect in oxygen metabolism and tissue respiration and for the purpose of maintaining near normal pulmonary ventilation.

5. It is pointed out that the carbon dioxide absorption technic was originally developed in anesthesiology to curb the tendency to hyperpnea that inevitably results when carbon dioxide in quantities over 3 per cent is rebreathed. The removal of carbon dioxide from the respiratory gases to an extent greater than 3 per cent is definitely deleterious to the welfare of patients undergoing major surgery.

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Gas Gangrene

REPORT OF AN UNUSUAL CASE, WITH A THERAPEUTIC PROGRAM FOR GAS GANGRENE

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THIS paper is presented as a brief review of the available data on gas gangrene, with special emphasis on the treatment of the disease. Physicians have been acquainted with this malady since Hippocrates first described the condition as an infection of the leg.[1]

TABLE I
ANAEROBIC BACTERIA IN GAS GANGRENE[3]

Organisms	Weinberg and Sequin	McIntosh	Henry
Cl. welchii	77	67	80
Cl. oedematiens	34	4	10
Cl. sporogenes	27	38	...
Cl. fallax	16	...	6
Cl. septicum	13	16	13
Total No. of cases	167	125	109

Peyronie in 1745 first distinguished between this disease and other infectious processes, and described the condition as gas gangrene.[2]

Etiology. The various species of clostridium are by far the most common etiologic agents, but occasionally other forms of bacteria may be responsible. Many tables listing the causative organisms are available,[1,2] and Table I by Trueta is fairly representative.[3]

Clostridium welchii (synonyms, Bacillus aerogenes capsulatus, Bacillus perfringens and Bacillus welchii) appears microscopically as a short, thick, encapsulated bacillus with truncated ends. It is surprisingly common, being abundantly present in soil, dust, sewage, milk, water, woolen clothing and fur of domestic animals. It is also common in the gastrointestinal and genitourinary tracts of man and animals. The individual characteristics of these bacteria are briefly described: Cl. welchii produce huge quantities of gas and several potent toxins, some of which cause hemolysis of red blood cells. Clostridium septicum is similar to Cl. welchii but is usually less severe. Clostridium oedematiens does not invade and dissect the tissues but produces symptoms, chiefly edema, by its toxins. Clostridium histolyticum produces tetany of the head and neck muscles but as a rule is in itself incapable of causing clinical gas gangrene.

TABLE II
ANATOMIC DISTRIBUTION OF GAS GANGRENE IN 607 CASES (MILLAR)[1]

Lower extremities	47.5%
Upper extremities	22.6%
Trunk and genitalia	16.3%
Undesignated areas	11.3%
Head and neck	2.3%

Incidence. In spite of the abundance of infective organisms present and a large percentage of traumatic wounds being contaminated with them, the clinical picture of gas gangrene is surprisingly infrequent. In a series of 187,936 such cases reviewed by Altemeier and Furste,[2] only 1.76 per cent developed clinical gas gangrene. The explanation for this is not completely understood. However, it is partly accounted for by a variation of virulence and by the fact that usually there is not enough impairment of the blood supply to provide the proper environment for progressive growth.

Gas gangrene occurs in various regions (Table II), the heavily muscled areas of the extremities being the most frequent site of involvement.

Pathology. Altemeier and Furste in their review[2] suggested the following classification:

I. Clostridial myositis
  1. Spreading or diffuse (gas gangrene)
    Crepitant
    Non-crepitant or edematous
    Anaerobic toxemia
  2. Localized myositis
    Crepitant
    Non-crepitant

II. Clostridial cellulitis (crepitant phlegmon or anaerobic cellulitis)

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Trueta describes the spreading or diffuse gangrene in the following manner:[3] (1) a central or dead zone consisting of disintegrated muscle destroyed by trauma, organized clot and a vast number of organisms; (2) a second or dying zone, which contains devitalized muscle fibers covered by masses of bacteria and products of infection; these fibers become separated by an exudate which may or may not be filled with leukocytes; (3) a third zone of relatively normal structures except for some cellular infiltration.

As a rule, the progress of the disease is as follows: The infection advances rapidly along the connective tissue planes and along the muscle fibers. When further extension is blocked by natural anatomic barriers, edema ensues; and as it steadily increases, the pressure produced gradually destroys the blood supply to the involved area, producing necrosis of the barriers. Once they give way, the infection once again advances. The involved muscle tissue becomes friable and hemorrhagic, and contracts producing a foul, watery, brownish exudate infiltrated with gas bubbles characteristic of gas gangrene.

It is important to remember that the infection has usually spread further than is clinically discernible. Examination of the wound usually reveals a dirty, dark area covered with the previously described exudate. The wound edges themselves may be black and the surrounding skin may appear tense, swollen and pallid. These areas become yellowish blue in color, and as the disease progresses, become gangrenous.

If the organism is Cl. oedematiens, gas may be absent and a widespread gelatinous edema may be present; marked intoxication may be a feature and some time may elapse prior to the appearance of gangrene. With Cl. oedematiens the muscle tissue has a dark red, raspberry color as contrasted to the Cl. welchii infection.[2]

Symptoms and Signs. Lazarus states that the average incubation period is about three days, but that it may vary from a few hours to ten years.[5]

Pain is usually the first symptom, becoming increasingly severe as time passes. This is accompanied by a rapid, feeble pulse which is out of proportion to a low temperature.

If an extremity has been the primary site of infection, the entire limb becomes edematous, engorged, discolored, cold and finally gangrenous. The muscle may herniate from the wound as an ugly gray necrotic mass neither contracting nor bleeding if cut.

As the disease becomes more severe, a peculiar grayish pallor, listlessness, weakness and profuse sweating develop. The patient then becomes dyspneic and his pupils dilate. In the terminal stages stupor, delirium and coma ensue, followed by the death of the patient.

Laboratory Data. The red blood count may be strikingly reduced to 1 or 2 million, the white count ranging from 12 to 15,000. Acidosis may be present.

Care should be taken in handling the smear and culture at the time of incision and drainage. Due to the infrequency of the disease, the laboratory should be carefully advised regarding the situation so that careful smears and cultures may be done.

DIAGNOSIS

As a rule the clinical diagnosis is easily made. The patient is critically ill, prostrated and apprehensive. There is persistent pain about the wound; devitalized muscle may be bulging through a wound with gangrenous edges, and thin, foul, watery discharge is usually present. Air crepitation is almost always present and is usually the diagnostic finding that points directly to gas gangrene.

X-rays may be quite helpful by demonstrating the air, particularly so when succeeding films show the gas to be moving along the fascial planes.

In situations in which the diagnosis is not clear, the following differential should be entertained:

1. Anaerobic cellulitis: This is a crepitant, septic process involving the connective tissue but rarely the viable muscle fibers. The patient's general condition is not critical and this disease is usually treated adequately by extensive drainage and antibiotic therapy.

2. Streptococcal myositis: This is a massive infection of muscle producing discoloration, edema, a serous exudate and gas. There is also considerable local pain and frequently marked generalized toxemia. It is obviously quite difficult to distinguish this from clostridial types of gas gangrene. However, there is a more pronounced cutaneous erythema, the muscle will respond to stimuli and is usually not necrotic. Finally, gram-positive bacilli can-

not be identified but, instead, large numbers of streptococci are present.

It should be noted that this disease is far more prevalent than is usually understood, being frequently observed in World War II.[8] Once the correct diagnosis is made by smear and culture, therapy consists of surgical incision and drainage as indicated and adequate antibiotic therapy. In the case report presented herein the patient was primarily treated as having a clostridial type of gas gangrene, but the culture showed a streptococcus.

3. Hemolytic streptococcal gangrene: This type of gangrene is predominantly subcutaneous with occasional sloughing of the overlying skin. Painful swelling and cellulitis is present, with a rather peculiar patchy necrosis of the tissues. Hemolytic streptococci are usually cultured with ease.

THERAPEUTIC REGIMEN

Indubitably, further antibiotic development will improve the course of therapy to be outlined, but the following basic principles should be adhered to:

Immediately upon confirmation of the diagnosis, antibiotic and antitoxin therapy should be begun while the arrangements for surgery are made.

Massive doses of penicillin should be given, preferably by intravenous administration up to 10,000,000 units per day.[6] It has been well substantiated by clinical observation and animal experimentation that the effectiveness is enhanced as larger quantities are employed.[10,11]

Aureomycin should also be started at this time by the intravenous method, and this may start at 6 gm. per day and the dosage lowered as warranted by clinical response.

Chloromycetin®[11] should be given orally (500 mg. every four hours) and this drug should also be reduced quickly. Intravenous chloromycetin is now available and should be used until oral chloromycetin is tolerated (1 to 2 gm. may be administered every six hours).[12]

The aforementioned antibiotics have been tested against the usual etiologic agents of gas gangrene and are believed to be the most beneficial.[13] Others are either ineffectual or are in the process of investigation at the time of this writing.

In reviewing the literature on gas gangrene antitoxin one intriguing fact is constantly encountered, namely, that the theoretic benefits have never been factually substantiated. Nevertheless, its therapeutic potentialities have been repeatedly demonstrated experimentally and this drug should always be administered immediately if the skin test is negative. If sensitivity to the horse serum is present, the drug should be started in small doses progressing as rapidly as possible.

This gas gangrene antitoxin (Lederle) contains the following: Cl. welchii, 10,000 units; Cl. septicum, 10,000 units; Cl. histolyticum, 3,000 units; Clostridium novyi, 1,500 units; and Cl. oedematiens, 1,500 units. In an acutely ill individual four of these vials should be given intravenously at once, followed by two vials every six hours until evidence of improvement is noted. The dose may then be reduced to one vial every six hours intramuscularly. Subsequent reduction of antitoxin should be carried out as dictated by the clinical picture. Such massive quantities of horse serum frequently cause serum sickness reactions and the patient should be observed carefully for this complication.

Surgery should be carried out as quickly as possible, and is still regarded as a most important therapeutic measure. This procedure is frequently carried out in the patient's room, thus confining the possibility of contamination to a small area. Current thinking leans toward a somewhat more conservative approach, particularly in the matter of amputation where an extensive incision and drainage may now be adequate. Nevertheless, the clinical findings usually dictate the scope of the procedure. Skin incisions should be extensive enough to allow exposure of all the underlying involved tissues, the error usually being that the incision is not inclusive enough or not through the fascial layers. If x-rays have been taken, they may well be useful here in demonstrating the extent of gaseous invasion. After the diseased tissues are adequately exposed, gauze fluffs saturated with hydrogen peroxide which has been mixed with zinc peroxide powder into a thin paste should be carefully packed against all the exposed surfaces. The entire wound should then be covered with a thin layer of xeroform gauze to assist in maintaining a high oxygen level. This dressing should be applied every six hours for the first forty-eight hours and then may be less frequent as improvement progresses. The area should be immobilized

without tension and care should be taken with casts.

If casts are being utilized, they should be split when applied; and if pain persists or if any doubt regarding the blood supply to the area is entertained, the cast should be removed and the wound again inspected. These patients are frequently in shock or preshock stages and blood should be used as indicated. The usual fluids and electrolytes should be employed, particularly watching for acidosis.

CASE REPORT

This forty-nine year old white man was admitted to the Bronx Veterans Hospital on May 7, 1950, following a brief illness. His symptoms began in December, 1949, and consisted of progressive right lower quadrant pain, increasing constipation and a 16 pound weight loss.

Physical examination revealed a hard, fixed, tender mass in the right lower quadrant. Hospital work-up pointed to malignancy of the ascending colon and on May 17, 1950, an exploratory laparotomy was performed. A large carcinoma of the ascending colon was present. This had perforated and a retrocecal abscess had formed, and the tumor had become adherent to the lateral abdominal wall. The regional lymph nodes were enlarged but no distant metastases were present. The carcinoma and regional nodes were removed, a right hemicolectomy being performed with an ileotransverse colostomy. An effort was made to excise the tumor completely from the parietal peritoneum of the lateral abdominal wall, but it was believed that the procedure was palliative. Beta streptococcus and Escherichia coli were cultured from the area. The patient recovered satisfactorily from the operation and was discharged on June 16, 1950.

On August 10, 1951, the patient was readmitted complaining of severe pain over the right hip which was initially interpreted as arthritic in nature. There was also a large hard mass present in the abdomen that represented recurrence of the original lesion. Two days following admission the patient fell on his right hip and this trauma relieved his symptoms temporarily. However, five days later the patient's temperature rose to 103.4°F.; the involved area became swollen and inflamed, and large amounts of subcutaneous gas were noted. A diagnosis of gas gangrene was made and the following measures were carried out. These steps have been described in detail and are simply enumerated here.

First, heavy antibiotic therapy was begun along with large amounts of gas gangrene antitoxin. Then the involved area was extensively incised and drained. A smear and culture were obtained and only hemolytic streptococcus was grown. It was believed at the time that this was a clostridial infection; however, this organism was never grown in the laboratory and it must be assumed that this patient represents a case of gas gangrene caused by a hemolytic gas-producing streptococcus. The wound itself was then carefully packed with gauze which had been saturated with a paste made up of a mixture of zinc peroxide and hydrogen peroxide. The intensity of treatment was gradually reduced as the patient improved. At the end of two weeks all therapy was discontinued except care of the wound. The area was then grafted and the patient discharged.

Shortly thereafter the patient returned to the hospital. He died rather suddenly on February 10, 1952. Autopsy showed generalized carcinomatosis. This growth was contiguous with the skin wound and the site of gas gangrene, and was thought to be entirely responsible for this complication which occurred approximately fifteen months following his operation.

SUMMARY

A basic, brief review of gas gangrene has been presented along with an unusual case. More important, a good, orderly therapeutic regimen has been offered for the treatment of gas gangrene.

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Crossett and Shaw have proven that with modern advances in medicine it is now possible to resect successfully diseased portions of the lungs of infants with relative safety. They performed pulmonary resections on six children under one year of age for bronchiectasis, chronic atelectasis, congenital pulmonary cysts or localized hypertrophic emphysema. They believe it is advisable to operate upon patients with bronchiectasis as soon as the disease is diagnosed, even in infancy, providing no special contraindication exists. (*Richard A. Leonardo, M.D.*)

Gas Gangrene of the Perineum

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GANGRENOUS periproctitis (gas bacillus gangrene) of the perineum is rare. The few references in medical literature are in textbooks on proctology.[1,2] Reported herein are two cases of gas gangrene of the perineum, with recovery. In one case the necrotic abscess was incised and drained in several stages. In the other case primary massive débridement and excision of involved tissues was completed in one stage; secondary closure by use of skin flaps and grafts was done later.

CASE REPORTS

CASE 1. A. E., an eighty-four year old man, was admitted on August 15, 1952, with the chief complaint of fever (103°F.) and diarrhea of three days' duration. Three months previously the patient had had an inguinal hernia repaired. Since then he had had loose bowel movements two to three times daily. Prolapsed painful hemorrhoids were present. There was no bleeding. Appendectomy had been performed in 1946 and a subtotal gastrectomy in 1949.

Physical examination revealed high fever, marked anxiety and severe toxicity. Abdominal wall incisions were well healed. There was an abscess (3 inches in diameter) in the left perineum about 2 inches from the anus. The skin overlying this abscess was blistered and edematous. Edema and induration extended into the left inguinal region and to the pubis. Several areas of gangrenous skin were present on the scrotum.

There was no improvement under conservative therapy for twenty-four hours. Softening and increased edema with crepitus were present in the perineum, left inguinal region, scrotum and at the pubis.

Under general anesthesia the perineal abscess was drained of a large amount of foul-smelling necrotic tissue. Bacteriologic examination indicated the presence of Clostridium welchii, other gas-forming anaerobic bacilli and Proteus vulgaris.

Drainage decreased and induration over the pubis increased. The skin over the scrotum which was not incised became gangrenous and spontaneously separated. On several occasions areas of induration were evacuated of a greenish, thick, purulent secretion. Anaerobic gas bacilli were again identified.

Local therapy included wet dressings of hydrogen peroxide and saline.

Operation was again performed ten days after admission, at which time devitalized tissue was excised. Applications of zinc peroxide suspension (Meleney[3]) were started. This was followed by considerable clinical improvement together with the appearance of new fringe areas of cutaneous necrosis.

A large amount of necrotic tissue separated spontaneously at this time. The testicles, the inguinal region and the perineum were denuded of skin. The area was exposed to dry heat using the perineal lamp. The areas improved slowly and granulation tissue appeared.

Eight weeks after the original operation a full-thickness skin graft was applied to the large defect in the inguinal region. This was 50 per cent successful. Two weeks later pinch grafts were placed on the perineum and inguinal area. Exuberant healing and granulation tissues continued to proliferate. Two months later all areas had healed. There was no further disability.

Antibiotic therapy used in this case was as follows: crysticillin® (300,000 units every eight hours), streptomycin (0.5 gm. every twelve hours) and terramycin® (250 mg. every six hours, orally). Eight vials of polyvalent gas gangrene serum were given during forty-eight hours.

Although blood sugar was 150 mg. per cent, there was no glycosuria nor was a diabetogenic curve observed on glucose tolerance test. One

month after admission blood urea nitrogen was elevated to 81 mg. per cent. Total proteins were low (between 5.2 and 5.7 gm. per cent). Parenteral feedings and blood transfusions were administered.

Comment. This patient with a large perineal abscess due to gas gangrene bacillus (involving the left inguinal region, left perineum, scrotum, both testicles and spermatic cords) was treated by multiple incisions and drainages supplemented by débridement. This was reinforced by antibiotic and antibody therapy. Two skin grafts were done. These did not fully succeed. Five months after original admission the area finally healed spontaneously. The patient is well and asymptomatic.

CASE II. E. S., a fifty-four year old man, was admitted to the hospital on January 13, 1953, because of swelling and pain in the left perianal region. This was of eight days' duration and had progressed slowly. Spontaneous discharge of a large amount of foul-smelling, dark green material had occurred two days prior to admission. There was some pain relief. Fever had been present for two days. Past medical history included weakness and anorexia during the previous year. The patient had been constipated and had taken milk of magnesia, cathartics and frequent enemas.

Examination revealed an afebrile, toxic, lethargic individual with a peculiar odor. In the left perianal region there was a sinus about 3 inches from the rectum. Foul, dirty green material was discharging from this sinus. The areas surrounding it were swollen, indurated, purplish and tender.

Overnight the inflammatory process spread rapidly. By noon of the day after admission the entire perineum, scrotum, left thigh and groin were swollen, indurated and purplish. Crepitation was observed. The blood sugar was 318 mg. per cent. Urine was strongly positive for sugar and acetone. Intravenous terramycin (2,000 mg.) and procaine penicillin were given. Insulin was started. By 4 P.M. he was semi-stuporous. Inflammation had extended to the apex of the left inguinal region, to the prepuce and the entire scrotum. A prophylactic dose of polyvalent gas bacillus antitoxin was given.

The patient was brought to the operating room where, under spinal anesthesia, the large gangrenous area was excised. The involved subcutaneous and superficial tissues from the apex of the left inguinal canal through the left groin and upper segment of the medial left thigh, the entire scrotum, the right groin up to the inguinal canal and the entire perianal region with the exception of the anal mucosa were removed *en bloc*. The rectal sphincters were exposed. The excised tissues were gas-filled, necrotic and foul-smelling with a creamy, greenish, serosanguineous exudate. It was necessary to excise perineal tissue back to the coccyx. Both of the left levator spaces were débrided and most of the left levator muscle was removed. The denuded area was washed with septisol® and packed with a freshly prepared, activated zinc peroxide solution on gauze. Rubber catheters were inserted in the dressings for continuous irrigation. A Foley catheter was inserted into the bladder for one week.

Following the last stage of this débridement the patient became unconscious and practically pulseless. He was revived by the administration of 50 per cent glucose (50 cc.), plasma (500 cc.), blood (500 cc.) and neo-synephrine®. His course thereafter was stabilized; he had no further evidence of shock or coma.

Postoperatively whole blood transfusions and parenteral fluids were given. In addition, he was given combiotic® (3 cc.) every eight hours. During the subsequent forty-eight hours he received four therapeutic doses of polyvalent gas bacillus antitoxin and 4,500 mg. of terramycin. On the fourth postoperative day oral feedings and oral terramycin were started. Intravenous terramycin was continued. Polyvalent gas bacillus antitoxin was given every other day for another week.

The wound was redressed every day and repacked with freshly prepared activated zinc peroxide. It was necessary to use trilene® analgesia[3] with the redressings.

One week later healthy granulation tissue was present. The wound was sprayed with furacin® solution every six hours and exposed to the heat lamp.

After the tenth postoperative day the patient was able to sit in a wash tub of hot water after each bowel movement. In addition, he irrigated the involved area by means of a shower head. Each bowel movement was comfortable.

The wound filled in rapidly. The general condition of the patient steadily improved.

Bacteriologic reports on the tissue at operation showed the presence of Cl. welchii,

Bacillus melaninogenicus together with Bacillus pyocyaneus and coliform bacilli. His diabetic status was rapidly controlled once the infection was overcome. Blood counts, urinalyses and blood chemistries were not otherwise abnormal.

On the twentieth postoperative day secondary closure[4] of the perineal wound was performed using a skin pedicle from the right thigh and complementary skin graft. A pedicle of skin was taken from the medial aspect of the right thigh, swung over the perineal body and attached to the lateral edge of the left inguinal canal. The defect at the origin of the pedicle graft was covered by a split-thickness graft. This healed well except for approximately 10 per cent of the distal edge of the pedicle graft. However, this area filled in rapidly with granulation tissue. The scrotal defect was approximated without any difficulty. Ten days later adjacent tissues were closed by transfer of a flap from the left thigh. The areas which were not covered by skin filled in rapidly. The patient was discharged from the hospital two months from the time of admission. Three months postoperatively all areas were healed.

Comment. The rapid onset of severe toxemia was unexpected in this case. At the time of hospital admission it was considered that the patient had the usual ischiorectal abscess. Perhaps bacteriologic study at the time of the original examination might have been valuable in saving a few hours. That recovery was rapid can be attributed in large part to the completeness in excision of all involved tissues, the adequate drainage and the efficiency of antibiotic as well as antiserum therapy.

Because of the presence of clean granulations and the absence of necrosis, early closure was accomplished to permit early rehabilitation.

SUMMARY

Two cases are reported in which patients suffered with gas bacillus infection originating in the perianal fossa and extending to involve the scrotum, genitalia, perineum, inguinal canals and thighs. In one patient incision and drainage was followed by staged d bridement and then by epithelization with aid of flap and pinch grafts. In the other patient primary débridement was accomplished followed by secondary closure using pedicle flap and split-thickness grafts. Antibiotics and antiserums were used in addition to surgical cleansing of the involved areas. Both patients recovered.

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Clinical Studies on Anticancer Agents from 1949 to 1951

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SEVERAL surveys have been conducted recently on *in vivo* experiments with anticancer agents.[137–139] The present study reviews some 190 clinical reports for the three-year period from 1949 to 1951 which have not been included in the aforementioned reviews.

ONIRGANIC AGENTS

Radiogold. Radioactive isotopes continue to be the most used inorganic chemical agents for the treatment of cancer. Müller has conducted extensive investigations on the use of radiogold. He employed balloons filled with the isotope on patients with carcinoma.[110,114] He also used intraperitoneal injections of colloidal radiogold against ovarian carcinoma.[112] Müller and Rossier[113] resorted to intravenous injections for patients with lung cancer. Müller and Held[115] treated lymph tumors by direct infiltration with colloidal Au-198.

Jentzer and Wenger[76] employed radiogold intraperitoneally in the treatment of carcinoma. Harsha[65] reported the isotope useful in palliation of fluid formation in neoplastic ascites and in lymph nodes.

Radiobromine. Smithers[153] used an intravesical bag to treat carcinoma of the bladder.

Radiocobalt. Müller[110,111,114] included Co-60 in his study of the therapeutic action of artificially radioactive elements on carcinoma of the bladder, rectum, uterus, etc.

Radioiodine. Carcinoma of the thyroid continues to be treated successfully with I-131.[110,114,153,155,181,182] Freedberg and Ureles[54] resolved lung lesions from adenocarcinoma with radioiodine.

Radiophosphorus. P-32 has been employed in the treatment of chronic leukemia,[13,108] leukemia in children,[125] lymphosarcoma,[153] carcinoma[110,114] and polycythemia vera.[153,181] Birge et al.[22] and Southam et al.[156] reported P-32 was without effect on acute leukemia.

Radiosodium. Na-24 was effective in patients with carcinoma of the bladder, etc.[153,181]

Radiozinc. Müller[110,113,114] carried out several studies on the use of Zn-63 in patients with neoplastic disease.

Antimony. Rubinstein[134] administered antimony intravenously in patients with multiple myeloma.

Arsenite. Florijn[53] used arsenite in the treatment of leukemia. Dameshek[36] treated polycythemia vera with $KAsO_3$.

ORGANIC NITROGEN COMPOUNDS

Nitrogen Mustards. Nitrogen mustards remain among the most important chemotherapeutic agents for the treatment of neoplasms. They have been administered successfully to patients with Hodgkin's disease,* adenocarcinoma,[7,17,19,88] lymphosarcoma,[2,9,11,102,147] leukemia,[3,8,11,25,132,178] and cancer of the lung,[6,11,84,96,133,178,186] skin[20,89] and liver.[21] Southam et al.[156] have reported that nitrogen mustards were without significant effect on acute leukemia.

Urethane. Urethane has found wide clinical application in the treatment of a variety of neoplastic diseases including leukemia,[28,33,53,78,93,129] polycythemia,[53] sarcoidosis[77] and mycosis fungoides.[82] Southam et al.[156] listed urethane as having little effect on acute leukemia. Lapides[90] also found the compound of no value in "androgen-independent" prostatic carcinoma. Urethane has been effective in multiple myeloma.[33,63,77,94,135]

Triazines. Triethylene melamine administered orally or intramuscularly has proved effective against leukemia,[62,79,106,121,192] lymphosarcoma,[79,80,107,131,146,192] Hodgkin's disease,[62,79,80,107,131,192] cancer of the breast,[62] fibrosarcoma,[192] and intra-arterially against tumors of the liver.[21] Trimethylolmelamine was effective against leukemia.[120]

Guanazolo. Straus et al.[165] reported guan-

* See references 2, 3, 6, 8, 11, 29, 33, 34, 46, 60, 102, 132, 147, 178.

azolo was ineffective when given intramuscularly in patients with Hodgkin's disease.

Sulfonamide. Bierman et al.[18] prolonged the life of children with lymphogenous leukemia with oral doses of sulfonamide.

Stilbamide. Propp et al.[127] recorded that stilbamide given intravenously relieved the pain of patients with multiple myeloma.

Purines. 2,6-Diaminopurine produced improvement in patients with acute leukemia.[24]

Pyridoxine Deficiency. Gellhorn and Jones[59] reported that a pyridoxine-deficient diet and desoxypyridoxine were without effect on lymphosarcoma.

β-naphthyldi-2-chloroethylamine. Matthews[100] recorded improvement of patients with Hodgkin's disease and myeloid and lymphatic leukemia following oral administration of this amine. Iverson and Meulengracht[75] found that five of six patients with polycythemia vera were improved, also.

p-Aminobenzoic Acid. Oral administration of aminobenzoic acid resulted in some improvement in patients with chronic myelogenous leukemia and lymphoblastoma cutis.[194]

FOLIC ACID ANTAGONISTS AND ANALOGS

Aminopterin. 4-Aminopteroyl glutamic acid is one of the most useful of all anticancer agents. It has found wide application in the treatment of leukemia.* Taylor et al.[174] considered this drug too dangerous for patients with metastatic cancer. Schoenbach et al.[141] gave this folid acid antagonist to patients with cancer of the lung and breast.

Amethopterin. 4-Amino-N^{10}-methyl pteroylglutamic acid has also been used extensively as an antileukemic agent.[24,52,104,105,136,161,184,187,191] Schoenbach et al.[141] reported this folic acid antagonist effective against neoplasms of the lung and breast.

Amino-an-fol. Wright et al.[191] administered this folic acid antagonist orally and intramuscularly to patients with leukemia, Hodgkin's disease, lymphosarcoma, etc. Farber[52] and Reinhard et al.[130] caused remission in patients with acute leukemia.

Pteroylglutamic Acid. Bichel[15] reported temporary remissions with PGA in an eighteen month old girl. Meyer et al.[103] caused remissions in leukemic patients with pteroyl triglutamic acid.

HORMONES

Androgens. Many reports have been made on the current use of testosterone in the treatment of cancer of the breast.* St. John[160] successfully treated papillary cystadenocarcinoma of the ovary with intramuscular injections of testosterone. Strohl[166] treated a patient with this hormone for metastasis of the femur. Lemon et al.[91] caused regression of adenocarcinoma of the thyroid with testosterone. Methyltestosterone has been used successfully in the treatment of cancer of the breast.[27,99,172] The use of androgens in neoplasms of the breast has also been recorded by Gardini,[56] Herrmann[68] and Nathanson.[117]

Estrogens. Stilbestrol has been employed successfully in the treatment of mammary cancer.[99,172] This estrogen has also been administered to patients with prostatic carcinoma.[5,55,116] Diethylstilbestrol is another estrogen which has been employed in cancer of the breast[30,31,38,69,74,101] and in prostatic carcinoma.[118]

Several reports have been filed on the use of estradiol in therapy of cancer of the breast.[31,61,172] Smith et al.[152] reported good results in the treatment of prostatic carcinoma with chlorotrianisene (TACE®). Two reports have been made on the administration of progesterone for prostatic cancer.[71,175] Smith also employed estrogens in patients with prostatic carcinoma.[151] Harrison et al.[64] combined estrogen therapy with orchiectomy in the treatment of prostatic cancer. Further reports on estrogen therapy for neoplasms of the breast have been made by Adair,[1] Garland,[57] Herrmann,[68,70] Lowenhaupt,[95] Nathanson,[117] van Winkle[176,177] and Walpole.[180]

ACTH. This miracle drug is finding increasing applications in the chemotherapy of neoplasms. It has been employed extensively in the treatment of leukemia,† Hodgkin's disease,[3,43,44,51] lymphosarcoma,[41,43,123,124,159,173] multiple myeloma,[43] lymphoblastoma,[163] mycosis fungoides,[171] beryllium granulomatosis,[83] adenoma of the pancreas[23] and carcinoma.[157,159]

* See references 12, 14, 16, 26, 32, 33, 35, 48, 52, 66, 73, 81, 97, 103, 125, 136, 148, 150, 161, 184, 187, 190, 191.

* See references 1, 4, 10, 27, 30, 31, 39, 47, 57, 61, 69, 86, 95, 98, 99, 101, 109, 126, 128, 144, 170, 172, 176, 177, 183.

† See references 3, 37, 40, 42, 43, 49–51, 67, 122–124, 142, 143, 154, 158, 159, 163, 164, 173, 188, 162.

Saunders and Adams[140] reported no effect of ACTH on leukemia.

Cortisone. This steroid hormone also has found wide usefulness in the treatment of leukemia,* Hodgkin's disease,[3,43,44,51,158] lymphosarcoma,[41,43,123,124,159,173] lymphoblastoma,[163] multiple myeloma[43] and carcinoma.[159]

Adrenal Cortex Extract. Saunders and Adams[140] recorded no effect of the extract on chronic lymphatic leukemia.

Pituitary. Wachtel[179] decreased the size of carcinoma and sarcoma in patients through subcutaneous injections of extracts of the posterior lobes of the pituitary gland of cattle.

Steroid Hormones. Emerson et al.[45] described the histologic alterations in cancer of the breast treated with steroid hormones.

MISCELLANEOUS

Podophyllin. Painting with podophyllin has been used in treating skin cancer.[85,168,189,193] Sims and Pensky[149] reported no appreciable clinical improvement following the application of this agent to basal cell epitheliomas. Sullivan[167] cured warts by applying podophyllintoxin.

Colchicine.® Levine[92] obtained arrested growth of carcinoma when patients were injected with colchicine.

Hyaluronidase Inhibitor. Kiriluk et al.[87] noted inhibition of neoplasms in patients given this enzyme inhibitor.

Penicillin. Bierman et al.[18] in their study of antibiotics and transfusions found that intramuscular injections of penicillin increased the survival time of children with lymphogenous leukemia.

Rabies Vaccine. Pack[119] noted improvement of a patient with skin cancer and another with a tumor of the liver when Harris' rabies vaccine was injected intramuscularly. Higgins and Pack[72] recorded eight definite regressions in patients with multiple metastases of melanoma treated w. h rabies vaccine.

Urine Extracts. Swann and Zelman,[169] by the parenteral use of extracts of urine from patients with chronic myeloid leukemia, caused temporary remissions in one patient with acute lymphoblastic leukemia.

Chymotrypsin. West[185] reported that intramuscular injections of this enzyme had no effect on the growth of lung carcinoma, lymphosarcoma and other neoplasms in fifteen patients. Shimkin et al.[145] reported negative results in ten cases of adenocarcinoma, melanoma, etc., following parenteral injections of this enzyme.

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* See references 3, 42–44, 49, 51, 123, 124, 142, 143, 154, 158, 159, 162–164, 173.

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POLLARD ET AL. recently surveyed thirty-eight patients with chronic duodenal ulcer upon whom a vagotomy (with or without gastroenterostomy) had recently been performed. About 85 per cent obtained satisfactory results. In previous years these same surgeons performed subtotal gastrectomy for chronic duodenal ulcers and obtained a similarly successful result in about 85 per cent of the cases. However, the vagotomy patients, a few years hence, may show a greater number of relapses, in my opinion. On the other hand, the mortality is apt to be much greater on the gastrectomized than on the vagotomized patients. (*Richard A. Leonardo, M.D.*)

Pulmonary Arteriovenous Fistula

A CASE REPORT WITH TREATMENT BY BASILAR SEGMENTAL RESECTION

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PHYSICIANS are becoming increasingly aware of pulmonary arteriovenous fistulas, as evidenced by the numerous diagnosed and surgically corrected cases which have appeared in the recent literature, but the actual incidence of this condition remains to be demonstrated.

Pulmonary arteriovenous fistula is not considered a disease entity in itself but is a part of a vascular defect presenting a varied pattern dependent upon the extent and location of associated vascular lesions. The disease complex has been referred to as Rendu-Osler-Weber's disease, hereditary hemorrhagic telangiectasis, and less often as cavernous hemangioma of the lung or familial telangiectasis.

From the earliest times[1–3] up to the year 1944, 1,000 cases of hereditary hemorrhagic telangiectasis have been reported.[4] The hereditary transmission of this disease had been referred to repeatedly and is generally accepted as fact.[5–11] Characteristically, multiple telangiectatic lesions of the facial skin, neck and lips, lesions of the mucous membranes, and lesions of the gastrointestinal tract as well as of various viscera occur. However, in none does it assume the striking form as it does when associated with a pulmonary arteriovenous aneurysm.[12–23] In this situation the triad of cyanosis, polycythemia and clubbing of the fingers and toes becomes evident.[3–6] In considering what modern surgical technic can offer the removal of a rather dangerous pulmonary lesion, each of the various forms of this condition presents separate and distinct problems.

In a review of pulmonary arteriovenous aneurysms in 1951 Blades and Beattie[25] reported at least fifty-three diagnosed cases, with thirty-three of the patients having undergone corrective operations. As recently as 1939 an antemortem diagnosis was first made of this condition. This was with the use of an intravenous contrast medium. The second antemortem diagnosis—the first to be cured by operation—occurred in 1942. A pneumonectomy was performed for lesions in the right middle and lower lobes.[12]

Patbology. A pulmonary arteriovenous fistula is essentially a direct communication between a distended afferent artery and a distended efferent vein, through one or several larger vascular trunks or a tangle of vessels instead of through the capillaries.[23] Although the pathogenesis is theorectic, reference is made to the plausible explanation offered in the common capillary plexus origin of arteries and veins.[26] Since there is absence of the muscle layer in the walls of the involved vessels, the probability of angioblastic activity and eventual fusion presents itself.[23] It is also possible that the absence of muscle is secondary to degenerative changes in the walls of the vessels.[27]

If pulmonary arteriovenous fistula is considered as part of hereditary hemorrhagic telangiectasis, it immediately becomes apparent that room to grow or expand exists most favorably in pulmonary tissue. It may be more proper to call these lesions arteriovenous aneurysms.

Patbologic Pbysiology. It is stated that 25 to 30 per cent of the blood volume must be shunted before cyanosis develops. The symptoms of this shunting, as seen in the third decade, are those of chronic anoxia.[3,8,28] There is polycythemia, an increased blood volume with a normal plasma volume and an increased hemoglobin concentration proportional to the degree of unsaturation of the arterial blood.[23] Sequelae of the polycythemia, manifested by thrombotic involvement of the smaller vessels of the central nervous system, give rise to symptoms such as vertigo, headache, convulsions, syncope, paresthesia, dysarthria or dysphagia,[27] making it difficult to exclude the possibility of the presence of an arteriovenous communication within the brain itself. The danger of a lethal pulmonary hemorrhage is always present.

* *Transferred to Letterman Army Hosp., San Francisco, Calif.*

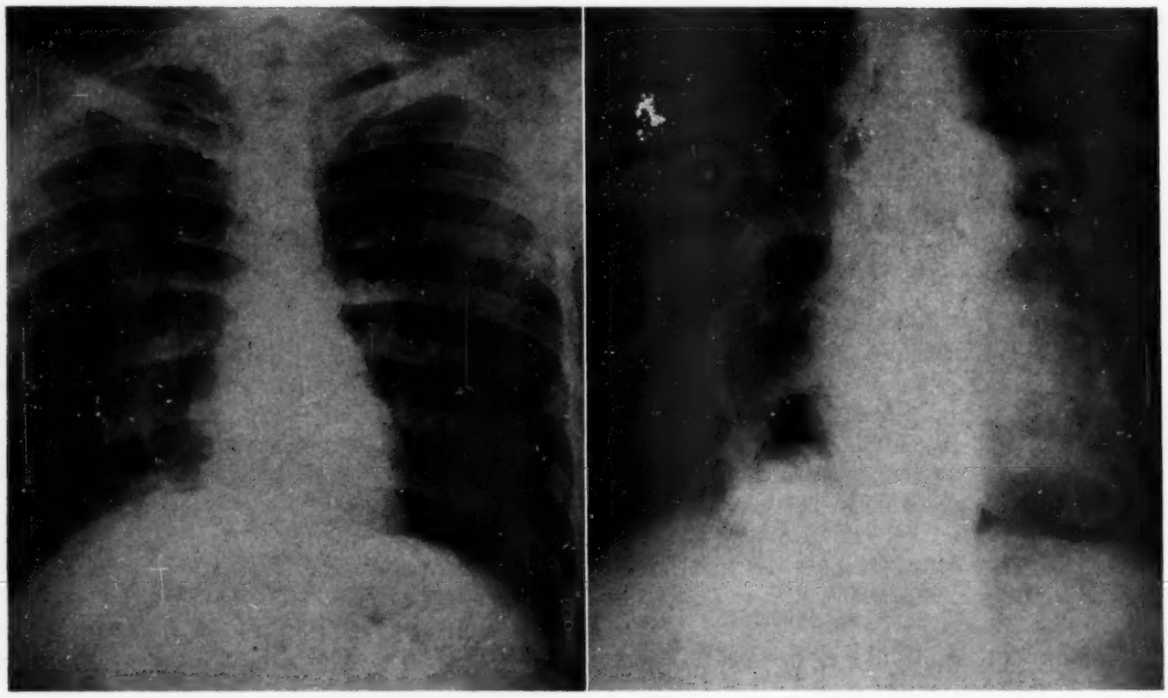

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FIG. 1. Postero-anterior x-ray of the chest showing the increased density between the right hilum and the cardiophrenic angle.

FIG. 2. Postero-anterior angiocardiogram showing the pulmonary artery entering and the pulmonary vein leaving the arteriovenous fistula in the right cardiophrenic angle.

Diagnosis. Cyanosis, clubbing and polycythemia, with or without telangiectases or hemangiomas of the skin or mucous membranes, are found in this condition.[25,27] A murmur is usually present directly over the lesion. It is loudest on inspiration and is continuous throughout the cardiac cycle. The heart is usually normal in size and without electrocardiographic changes.

Roentgenograms will usually show an increased density continuous with the vascular shadows of the hilum. These densities, however, may be multiple and bilateral. An absolute diagnosis of the lesions in the lung found radiographically depends on angiocardiography.

Fluoroscopically, the mass in the lung may be seen to pulsate with the cardiac cycle and is altered in size.[23]

Differential Diagnosis. An aneurysm of the pulmonary artery must be ruled out. The x-ray appearance is somewhat similar, but this lesion is quite rare and blood studies are usually normal in this condition. In addition, there is no cyanosis or clubbing of the fingers. Ordinarily congenital heart disease can be ruled out by physical examination and other diagnostic procedures. Absence of basophilic stippling and immature white cells will distinguish polycythemia vera from pulmonary arteriovenous aneurysm. Metastases, adenomas, cysts and tuberculomas must all be differentiated.

Singer and Wolfson,[29] in a very fine analysis of the common distinguishing features of hereditary hemorrhagic diatheses, pointed out that with a normal bleeding time and a negative tourniquet test hereditary hemorrhagic telangiectasis is present in its purest form. If positive tourniquet tests have been reported, a search should be made for the coexistence of pseudohemophilia or hereditary familial purpura. In addition, platelet alterations, as in thrombocytopenic purpura or thrombasthenia and other disturbances of the hemostatic mechanisms such as hypofibrinogenemia and hemophilia, are usually present in the differentiation of the hereditary hemorrhagic diseases.

Treatment. The treatment of pulmonary arteriovenous aneurysms is surgical to obviate the hazard of a future lethal pulmonary hemorrhage or thrombotic episode due to the polycy-

themia. Conservation of as much lung tissue as possible is of prime importance since these lesions are apt to be multiple and bilateral.

CASE REPORT

A twenty-three year old white man was admitted to an Army hospital after twelve hours of hemorrhage following the extraction of teeth. He was subsequently referred to Walter Reed Army Hospital for definitive therapy.

There was a history of cyanosis, dyspnea on exertion with associated chest pain, difficulty in swallowing, headaches lasting up to a week unrelieved by aspirin, and blurring of vision in intermittent attacks since childhood. Four to five months before admission he had aching pains in his legs and knees, especially while walking.

The patient's mother had been cyanotic for as long as he could remember. She also had frequent attacks of epistaxis without associated trauma. A maternal uncle had episodes of epistaxis and "bleeding from the eyes." The patient's brother had a tendency to bruise easily and as a child had had episodes of epistaxis without cause.

On physical examination there were scattered, small, telangiectatic lesions on the skin of the upper and lower lips, chest, hands, back of the neck and on the nasal septum. Auscultation of the chest revealed a continuous murmur over the right posterior base accentuated with systole and inspiration. There was clubbing of the fingers and toes with cyanosis of the nail beds.

Chest films and fluoroscopy showed a slight increase in the vascular markings at the right hilum. These markings were continuous with a globular mass posteromedially in the right base. (Fig. 1.) There were pulsations of this mass, as well as alterations in its size with Valsalva and Mueller maneuvers, demonstrable fluoroscopically. Rapid sequence angiocardiography demonstrated a filling of the pulmonary artery first, then the mass, and immediately thereafter the pulmonary veins, so that on the last film all three structures were visible. (Fig. 2.) At the time of admission to Walter Reed Army Hospital hemoglobin was 17.9 gm. with a hematocrit of 54 per cent. The red blood count was 6.05 million; bleeding time, 7 minutes; coagulation time, 3 minutes; platelet count, 486,460. Esophagoscopy and gastroscopy were negative for any abnormality. Stool specimens were negative for blood.

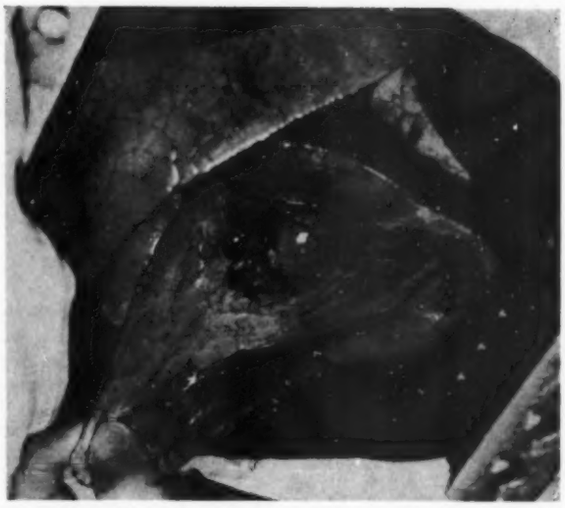

FIG. 3. Photograph of pulmonary arteriovenous fistula at operation showing the fistula on the diaphragmatic surface of the right lower lobe.

At operation on May 15, 1952 segmental resection of the basal segments of the right lower lobe was performed with complete extirpation of the fistula. (Fig. 3.) The patient's postoperative course was complicated by a bronchopleural fistula which responded to closed thoracotomy drainage. The postoperative hemogram returned to normal with a red blood count of 4,650,000 and a hemoglobin of 13.1 gm. The cyanosis completely disappeared. Tourniquet tests performed pre- and postoperatively were within normal limits. After complete clearing of the lung fields the patient was discharged to full duty.

SUMMARY

The pathology and pathologic physiology involved in pulmonary arteriovenous fistula is reviewed and a case presented in which extirpation of the fistula was accomplished by basilar segmental resection. The treatment of this condition is surgical with conservation of as much lung tissue as possible.

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Dissecting Aneurysm of the Aorta and Its Relation to the Acute Condition of the Abdomen

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DISSECTING aneurysm of the aorta is not generally considered as lying within the realm of the surgeon and consequently is not often kept in mind in the differential diagnosis of abdominal emergencies of puzzling etiology. Nevertheless, it occurs with sufficient frequency and the surgeon should always remain aware of its existence. Needless surgical exploration may thus be avoided. Although the majority of dissecting aortic aneurysms are treated by the internist because the process is confined to the chest, a substantial number of them produce abdominal pain as the outstanding symptom (about 25 per cent in one series of fifty-eight cases). Consequently, the surgeon may be called to deliberate on the presence of an acute surgical condition of the abdomen. Dissecting aneurysm may mimic any abdominal catastrophe. I believe most surgeons of experience can subscribe with chagrin to the truth of this statement. Syndromes resembling acute pancreatitis, acute appendicitis, perforated ulcer, mesenteric infarction, peritonitis, ureteral colic, etc., are not unusual. The diagnosis unfortunately is seldom clear and an antemortem diagnosis is a rarity. In this respect it may be well to point out that only thirteen correct antemortem diagnoses were made as recorded in the world literature up to 1937. This figure, I venture to say, is probably not very much higher now.

Pathology. Dissecting aneurysm is not a true aneurysm but the result of the forcible cleavage of the walls of the vessel by blood. Despite much theorizing the true cause is not known. It is now generally believed that such conditions as atheroma, arteriosclerosis, lues or intimal injuries cannot be indicted. The basic cause seems to lie in the media of the vessel wall. Localized necrosis of this layer results in the formation of cystic spaces of mucoid material. Whether the production of this mucoid material is caused by or is the result of disintegration of the elastic fibers of the media is not clear. At any rate this weakness of the media is usually present, and any rise in blood pressure high enough to rupture the intima over such a debilitated area seems sufficient to initiate the flow of blood into the cystic media for the evolution of a dissecting aneurysm.

Any sudden increase in blood pressure in a hypertensive patient, due to any cause (such as sexual intercourse, work, excitement), in the presence of a diseased media may be sufficient to cause perforation of the intima. Dissection of the vessel wall proceeds within the media and may continue for considerable distances. This process thereby often reduces or obliterates the natural lumen by pressure extrinsic to the intima. Consequently, marked vascular disturbances result, with ischemia or even necrosis of the organs supplied by the involved vessel. Usually external rupture through the adventitia produces a lethal hemorrhage. In about 10 per cent of the cases, according to some authors, the dissecting channel may burrow through the intima at another higher or lower level and the blood re-enter the vessel. In time the false passage may then become obliterated and healing may take place. The extent of the dissection seems to bear no relation to the survival time. Indeed, extensive dissection may be quite compatible with life as long as there is no break through the adventitia.

Incidence. The frequency of this disease is difficult to assess. Dissecting aneurysm has been reported at both extremes of age. The onset of dissecting aneurysm during the course of pregnancy has been notably high. However, it seems to occur most predominantly in males forty to sixty years of age. With the progressive

longevity of our population there is no doubt that it will be met more frequently in the future. The largest series reported to date has been fifty-eight cases. It may be of interest that in a series of 8,200 autopsies reported at the Massachusetts General Hospital, dissecting aneurysm was noted once in every 430 autopsies. Unfortunately, many cases of sudden death are not subjected to autopsy. Many of these may be due to dissecting aneurysms and certified as deaths from other causes such as coronary disease (its most prevalent imitator). Higher autopsy percentages would unquestionably establish a higher rate of incidence of this disease than is presently accepted.

Diagnosis. Even though there is a much greater awareness of this condition at the present time, a correct antemortem diagnosis is still noteworthy. There are no typical findings; and because the signs and symptoms are so varied and confusing, diagnosis rests largely upon the acuity and intuition of the examiner. The point has been made that every patient with acute abdominal pain who also has high blood pressure should be suspected of having a dissecting aneurysm as well as an acute condition of the abdomen. It should be considered in any acute abdominal emergency of puzzling etiology. Presenting a long list of differential diagnoses is of no value in clarifying the picture. Any abdominal emergency may be simulated since the vascular supply of any abdominal structure may be shut off or impaired by this process. Ischemia of organs is probably the fundamental cause of most symptoms before actual aortic rupture takes place. Subsidence of pain should not lull the observer into relaxing his vigilance since recurrence of pain is common in patients surviving the initial episode. Renewed pain indicates either further dissection or adventitial rupture.

Findings. There is a sudden onset of pain anywhere in the abdomen and frequently referred to the back or into the legs. The severity of the pain and the associated symptoms of course depend entirely upon the degree and rapidity of effusion of blood into the extravascular tissues and therefore may be extreme and agonizing or may be only a moderate ache. The distribution of the pain likewise depends upon the course taken by the intravascular dissecting channel, the vessels and organs involved, the direction and degree of extravascular hemorrhage or the degree of ischemia resulting from the compression of the main lumen by the mass of blood in the new channel. Seepage of blood into the abdominal cavity produces symptoms of peritoneal irritation. Ischemia and anoxia of the structures deprived of blood is plausibly the main source of pain in those patients who do not immediately succumb to an exsanguinating extravascular hemorrhage.

Vomiting occurs quite commonly. Moderate fever and leukocytosis are usually present and generally are of no diagnostic significance.

Rapid pulse and moderate or marked cardiac enlargement are usually noted. An electrocardiogram is always of value in that it helps to rule out acute myocardial infarction as the cause of the presenting syndrome. Left ventricular hypertrophy is a common observation.

A characteristic finding is said to be loss or diminution of the pulses in one or both legs and a marked blood pressure differential between the upper and lower extremities.

Prostration and shock are marked. However, the blood pressure remains well above the shock level. It is to be remembered that most of these patients have a history of hypertension, usually of long duration. Furthermore, various authors have stressed that even though the patient may have a moderate or high pressure upon examination, the blood pressure may well have been considerably higher before the onset of the present catastrophe and that this pressure may be at the shock level for this patient, while for another patient it might be considered normal or above normal limits.

Mortality. Ninety per cent of patients with dissecting aneurysm die within a week of its inception, the majority within twenty-four hours. A few are said to recover and the aneurysm may heal. Occasionally re-entry into the lumen may take place and the false passage may become obliterated.

Surgical Treatment. Most patients do not live long enough for a diagnosis to be established. There is mention of surgical intervention in dissecting aneurysms of the iliac vessels whereby re-entry of the dissecting channel into the natural lumen was performed. These reports are scarce and the results presumably discouraging. There is no plausible reason why some of these aneurysms should not be amenable to surgical correction by the same means employed today for other aneurysms. Aortography should be of value in the

diagnosis and pinpointing of the lesion in cases of gradual onset or prolonged survival time. No such studies to my knowledge have been reported to date.

CASE REPORTS

CASE I. . A seventy-three year old white man was admitted to the hospital with a history of generalized abdominal pain and vomiting of twenty-four hours' duration. His temperature was 101°F. on admission and then rose to 103°F. Leukocytosis of 29,000 was present. The urine contained albumin and hyaline casts. Blood pressure was 180/120. At the time of admission this patient had spasm of the entire right abdomen. Tenderness was marked over McBurney's point and somewhat less pronounced over the right hypochondrium. Rebound tenderness was present in both locations. The examining surgeon thought that the patient had acute appendicitis but, because of the age and surgical risk involved, called for medical consultation. The medical consultant ordered an electrocardiogram which showed evidence of myocardial damage and left ventricular hypertrophy but did not disclose coronary involvement. Considerable cardiac enlargement was noted. The medical consultant made a diagnosis of acute cholecystitis. Before any treatment was instituted, the patient died suddenly. Autopsy disclosed a normal abdomen with a gallbladder packed with stones. A large retroperitoneal hematoma was found and the cause of death established as a ruptured dissecting aneurysm of the upper abdominal aorta with dissection into the right renal artery. The rent in the aorta was about 3 inches long. There was no extravasation into the chest.

CASE II. A fifty-eight year old obese man was admitted to the hospital because of fever and severe pain in the back and right upper abdomen. He had been treated at home for one week for a "virus infection" without relief. Three years previously the patient had been treated for what was believed to be a right ureteral calculus. He had had hypertension of 180 systolic for several years. A surgical consultant found the patient to have a temperature of 102°F. The white blood count was 22,000. The urine showed casts and red blood cells. There was marked tenderness over the right kidney. An intravenous pyelogram did not visualize the kidney pelves. The surgeon believed that the patient had no surgical condition of the abdomen, and requested a genitourinary consultation. The urologist ordered the patient readied for cystoscopy the following day. In the meantime fever and pain had subsided and the patient wanted to go home. He was prevailed upon to stay. That night he suddenly went into collapse and was dead in a matter of minutes. Autopsy revealed a massive retroperitoneal hematoma. A ruptured dissecting aneurysm of the lower thoracic and upper abdominal aorta was found, with dissection into the right renal artery. The rent in the aorta was 7 inches long.

In both cases histologic study showed typical focal necrosis with mucoid transformation of the media.

SUMMARY

There is no intent in this paper to review the entire subject of dissecting aneurysm, which has been fairly well covered in numerous articles. Most cases are treated by the internist. However, there is a surgical problem. It is in those patients with abdominal symptoms that the surgeon is confronted with a diagnostic dilemma. Presumably, one of four patients with dissecting aneurysm of the aorta may present signs and symptoms indicating an abdominal surgical emergency.

All patients with hypertension who present evidence of an acute surgical condition of the abdomen of doubtful etiology should be suspected of having a dissecting aneurysm or aortic rupture.

Two recent case reports have been added for illustration. Both patients entered the hospital within a few weeks of each other and their hospital course stimulated this paper.

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Mixed tumors of the parotid, serous cell adenomas, benign mucoepidermoid growths, papillary lymphoid cystadenomas and acidophilic cell adenomas, classified as benign by Buxton et al., require in their therapy only thorough local excision with preservation of the facial nerve. Malignant lymphoepitheliomas may be benefited by roentgen therapy but the other malignant tumors, especially the squamous and gland celled carcinomas, should be submitted to radical excision of the parotid including its imbedded facial nerve, and often also including a radical dissection of the glands in the neck. (*Richard A. Leonardo, M.D.*)

Mesenteric Chylangioma

WITH REPORT OF TWO CASES

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MODERN textbooks of surgery and pathology make little or no mention of chylangioma or chylous cysts, a fact which is indicative of the rarity of this occurrence. Block[5] writes that they occur once in every 150,000 hospital admissions. Hence it is evident that mesenteric cysts are sufficiently uncommon to arouse considerable interest, for they are the rarest of abdominal tumors. Their incidence is best illustrated in Table 1.

A chylous cyst of the mesentery is any cystic tumor that contains a milky fluid resembling chyle in appearance.

Warfield[21] emphasized that many surgeons of wide experience have neither observed nor operated upon a patient with a mesenteric cyst. The consensus is that the condition occurs twice as frequently in women and it is rarely found in colored races. Of all the mesenteric tumors, the cystic variety is four times more common than the solid type. The cysts are usually diagnosed in early adult life, although they have been found in children. In fact, mesenteric cysts have been found at all ages, in the fetus and in the octogenarian. However, Benedict[4] is of the opinion that no age is especially predisposed to abdominal cysts.

TABLE 1
FREQUENCY OF MESENTERIC CYSTS

Surgeon	Hospital	Admissions	Cases of Cysts
Costello	Women's and Children's Hospital, Toledo, O.	28,312	1
Judd and Crisp	Mayo Clinic, Rochester, Minn.	820,000	8
Kanski	Mercy Hospital, Buffalo, N. Y.	135,743 (25 yr.)	2
Loeb	Bronx Hospital, New York, N. Y.	105,000	1
Olesen	Los Angeles Hospital, Los Angeles, Calif.	(1912–1929)	0
Porter	St. Mary's and St. Luke's Hospitals, Duluth, Minn.	200,000	2
Porter	University of Minnesota Hospital	15,000 autopsies	0
Roller	University of Pennsylvania	900 surgical (1931–1933)	3
Roller	University of California	93,511	1
Roller	Children's Hospital, Los Angeles, Calif.	12,425	3
Roller	General Hospital, Los Angeles, Calif.	188,921	1
Vaughn et al.	Mercy Hospital, Chicago, Ill.	750,000	3
Roller	Massachusetts General Hospital, Boston, Mass.	(1900–1926)	6

CASE REPORTS

CASE 1. G. L., a twenty-eight year old white woman, was admitted to Mercy Hospital on September 3, 1951. Her chief complaint on admission was that of low abdominal cramp-like pain, as well as a similar type of pain in the lower back region, over a period of two or three months prior to admission. She had felt a tumor in the left side of the abdomen for the past two months, which was not tender but seemed to increase in size; she had no loss of weight; bowels were regular.

The patient was gravida 1, para 1. Her first admission was in November, 1949, when surgery was performed, namely, partial right oophroectomy, removal of paroöphoron cysts (six) right, biopsy of node of broad ligament, appendectomy and Coffey hysteropexy. Her past history at that time included two previous operations for the removal of tumors of the female generative system, type unknown. Her next hospitalization at Mercy Hospital was in April, 1951, at which time x-ray examination revealed multiple cysts of the mandible. Admission diagnosis was possible adenoma of the pituitary, but x-ray examination of the sella turcica revealed a generally normal pattern of trabecular structure and vascular grooves in the bony calvarium. The sella turcica was of the closed type, small, but had

no evidence of osteolytic or erosive involvement in its surrounding cortical structures.

Physical examination revealed a well developed, well nourished patient who was alert and in no apparent distress. Blood pressure was 120/80. Head and neck were normal. The sternum was depressed below the manubrium with a "pigeon breast" deformity. Breasts were large and pendulous; there were no masses. Heart and lungs were normal. The abdomen showed scars from previous operations. A freely movable mass was felt in the left hypochondrium which was tender on deep pressure. There was a well healed Pfannenstiel scar.

Laboratory data were as follows: Blood count showed white blood cells 10,000; hemoglobin, 13.5 gm., 87 per cent; red blood count, 4,500,000; polymorphonuclears, 74 per cent; basophils, 1 per cent; lymphocytes, 25 per cent. Serology showed no reaction. Her blood type was AB, Rh negative.

At operation a left paramedian skin incision was made beginning to the left and above the umbilicus 2 inches and extending downward. On entering the peritoneal cavity it was found that the omentum was adherent throughout the anterior abdominal wall and the previous upper abdominal incision. This was freed by sharp dissection. All bleeding points were tied. Several adhesive bands involving the jejunum were freed and the root of the mesentery was exposed which showed a baseball-sized, white, smooth tumor present in the leaves of the mesentery near the root; there were also three similar smaller sized tumors. No free fluid was present. Two of the smaller tumors were entered on attempting to remove them, and a milky substance presented itself. The remaining portions of these tumors were removed. A smaller tumor was exposed and removed intact, as was the larger tumor, after splitting the mesentery. The vascularity of the jejunum was examined to make sure that the circulation was not disturbed in removing this large-sized tumor. The color was good and the cut edges of the mesentery were closed with interrupted silk.

The incision was closed in layers; posterior rectus sheath and peritoneum with continuous chromic sutures; anterior rectus sheath with interrupted wire; skin with skin clips and two interrupted dermal stays.

The postoperative diagnosis was abdominal adhesions, chylous cyst, root of jejunal mesentery.

Postoperative treatment was as follows: 1,000 cc. beclysl® and 1,000 cc. 5 per cent glucose solution intravenously; duracillin,® 400,000 units every twelve hours; 1,000 cc. amigen first day postoperatively.

The patient had no complications following surgery. She was emotionally upset the third postoperative day. On discharge the eighth day following surgery, the incision was clean; all clips were removed.

Pathologic report revealed the following: Gross specimens consisted of a firm nodule 5 by 4 by 4 cm. On section it appeared to be a cystic structure filled with cheesy, pale yellow material. The second nodule received measured 1½ cm. in diameter and was also filled with creamy, cheesy material. A third specimen was a lentil-sized nodule 4 mm. in diameter.

Microscopically, section through both cysts showed a thick fibrous wall infiltrated by chronic inflammatory cells. The inner surface appeared to be lined by an endothelium. Adherent to the surface were pink debris and foamy cells containing lipoid. The inner wall also showed lipoid resorption. One of the sections showed a portion of lymph node in the wall of the cyst. Altogether the picture was that of chylous lymphatic cysts of the mesentery, nothing malignant.

Case II. G. L., a thirty year old white woman, was admitted to Mercy Hospital on August 23, 1953. Her chief complaint on admission was that of low back pain of about one year's duration. She also had a tumor mass of the abdomen to the left of the navel, six months' duration, tender. The patient stated that she felt nauseated when she pressed on it. Bowel movements were irregular and there was no history of tarry stools, weight loss or other complaints.

Patient was gravida 1, para 1. She had had rickets during childhood. Her first operation was in 1942, for a tumor of the stomach. A similar operation had been performed in 1951 (tumor of the abdomen). In 1949 she had been operated upon for an ovarian cyst.

Physical examination revealed a fairly well developed, fairly well nourished patient, with acromegalic facies, who was coherent and up and about. Blood pressure was 110/64. The head and neck were normal. Patient had funnel-shaped deformity of the sternum and upper ribs. A palpable mass the size of a "tennis ball" was present in abdomen to the left of umbilicus

—freely movable and tender. The patient had a well healed Pfannenstiel scar and left paramedian scars of the abdomen.

Laboratory data were as follows: Urinalysis was negative for albumin and sugar. There were 3–5 white blood cells per high-power field. Occasional epithelial cells were noted. Blood count showed white blood cells 4,850; hematocrit, 41 per cent; segmented neutrophils, 51 per cent; eosinophils, 4 per cent; monocytes, 7 per cent; lymphocytes, 38 per cent. Serology showed no reaction. Her blood group was AB, Rh positive.

Radiographic examination of the abdomen revealed a poorly defined area of increased density obscuring the lower portion of the left psoas shadow and kidney border and appearing to exert an extrinsic pressure defect on the medial border of the descending colon; the possibility of a soft tissue mass in the region of the surgical scar was considered.

At operation a chylous cyst of the mesentery was removed. Excision of a previous, left paramedian skin incision was made, exposing the anterior rectus sheath which was cut. The rectus muscle was split and the posterior rectus sheath and peritoneum were cut one-half medial to the skin incision. On entering the abdominal cavity, a very large grapefruit-sized, smooth, milky mass presented itself at the mesentery of the jejunum, near the ligament of Treitz. The adjacent 16 to 18 inches of jejunum was markedly congested, cyanotic, apparently due to pressure of the mass on the intestinal vessels. Some adhesions were present between the loops of the jejunum. These were freed and the jejunum together with the involved mesentery was delivered into the operative area; because of the marked involvement of the mesentery down to its root with this tense, smooth, milky mass, a resection of the involved mesentery and jejunum was performed, removing approximately 18 inches of jejunum at least 3 inches medially and laterally to the involved mass. The entire mesentery involved was removed without disturbing the cystic mass. A side-to-side anastomosis was performed; the distal loop overlying the jejunum approximately 4 inches distal to the ligament of Treitz. A two-layer technic enclosure was employed, the inner hemostatic layer taking in all three layers of bowel with No. 0 chromic suture. The serosa was closed with interrupted Lembert suture. Both angles of the anastomosis were reinforced with interrupted silk. Examination of the rest of the mesentery revealed an additional tangerine-sized, smooth, yellowish cystic mass, approximately 6 inches away from the resected mass. This was removed by enucleation. The rent in the mesentery was closed with interrupted silk. No additional masses were seen or felt. There were some fine adhesions between the loops of the jejunum which was otherwise quite clean. The incision was closed in layers without drainage; peritoneum and posterior rectus sheath with interrupted chromic suture; anterior rectus sheath with the same; skin closure with skin clips and interrupted dermal stay. A transparent plastic type of dressing was employed. No gauze was used.

The postoperative diagnosis was chylous cyst. Postoperative treatment included active Wangensteen suction for three days. After surgery 1,000 cc. of whole blood were administered. Also 1,000 cc. of beclysl with alcohol and 1,000 cc. of trinidex® were given. Pantopon® gr. ⅙ p.r.n. for pain, 250 mg. of streptomycin and 400,000 units of penicillin were administered every eight hours; vitamin K, 20 mg. every four hours; 1,000 cc. of 10 per cent travert® solution; solu B,® 2 cc. given intramuscularly; and ascorbic acid, 250 mg. every six hours.

On discharge the eighth day following surgery, incision was clean; all clips were removed.

Pathologic report was as follows: Gross examination revealed a loop of small bowel 35 cm. in length, of normal caliber; the lumen was empty. The wall of the bowel was slightly edematous but the mucous membrane was not remarkable. In the mesentery there was a nodular cystic mass 8 by 7 by 6½ cm., with a lobulated surface, and two other cherry- and walnut-sized nodules which appeared yellow and cystic and contained creamy thin fluid. On section through the larger mass, it appeared to be a thick-walled cystic structure. The wall was very fibrous, and averaged about 8 mm. in thickness. In the wall of the large cyst there were several pea-sized daughter cysts containing creamy fluid. (Fig. 1.)

Microscopically, section through the bowel showed moderate edema of the wall; otherwise it was not remarkable. Section through the many cystic structures from the mesentery showed a varied picture of thin and thick wall cysts. Most of the cysts were lined by endothelium; some showed distinct chronic inflam-

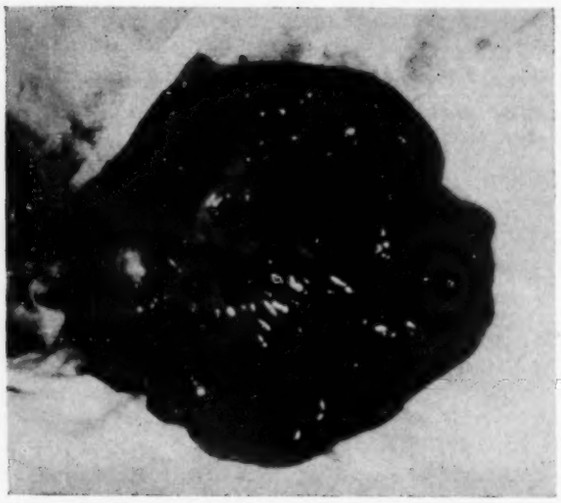

FIG. 1. The specimen removed in Case II. Note the smooth tumor mass in the mesentery of the jejunum.

mation in the wall and evidence of lipoid resorption. In addition, many had masses of nodular lymphatic tissue in the wall resembling lymph nodes. On the outer side of the cysts there were many dilated irregular lymphatic channels. Section through several of the lymph nodes in the mesentery showed a moderate chronic non-specific reactive inflammatory hyperplasia. A few of the nodules, thought to be lymph nodes, had a peculiar makeup of dilated lymphatic channels, loose primitive connective tissue and masses of lymphoid tissue; this picture suggests a lymphatic hamartoma. Altogether, the anatomic changes were those of cystic dilated lymphatic channels filled with chylous fluid and many dilated lymphatic irregular vessels.

COMMENTS

Benevieni, the Florentine anatomist, in 1507 was the first to observe mesenteric cysts, calling them "anatomic curiosities." Rokitansky, in 1842, published the first accurate description of a chylous cyst found at necropsy. Plan, in 1883, reported the recovery of a patient with a chylous cyst treated by marsupialization.

Chylous cysts, cholangiomas, as some refer to them, are usually multilocular and occasionally occur as numerous small swellings of the mesentery, intestines, omentum or retroperitoneal organ, according to Thompson and Chambers.[19] They are more common in the mesentery of the ileum and jejunum than that of the colon. They may be simple or multiple, unilocular or multilocular, and may vary in size from that of an olive to enormous growths filling the abdominal cavity.

Chylous or chylangioma cysts are thought by some to arise by obstruction of a lymphatic trunk. Ladd and Gross[11] doubt this theory because of the absence of demonstrable inflammation or other fibrosing lesions in the mesentery. They believe that these cysts develop from misplaced bits of lymphatic tissue which proliferate and then accumulate fluid because of lack of a communication with the remainder of the lymphatic system.

It has been suggested that chyle-like fluid may come from connections with chyle-bearing lymphatics or through fatty degeneration of the tissue lining these cysts (foam cells). This reasoning follows since chylous cysts of the mesentery are found associated with the chyle lacteals of the mesentery or the thoracic duct in the retroperitoneal space.

Block[5] states that the source of chyle in these cysts has not been definitely determined, while Ewing[7] believes that true chylangiomas are due to congenital or acquired obstruction of lacteals. Beahrs, Judd and Dockerty[3] assert that mechanical obstruction to the lymphatic vessels must be discounted since the cysts have not been produced by experimental obstruction of the thoracic duct and because of the extensive anastomosis of the lymphatic vessels. Lee, Ewing[7] and others think of trauma as a possible cause in some cases, while Dowd[6] and his followers believe they are developmental anomalies. Loeb[12] is of the opinion that aberrant thyroid, ovarian, splenic or liver tissues may be found anywhere in the body. They may also migrate and remain between the layers of the mesentery. The intervals between the layers is reported to be filled with fifty to 150 lymph vessels, the superior mesenteric artery and its numerous branches that supply the small intestines. Lymph glands, as well as varying amounts of fatty tissue are also found in this mesenteric space. As the layers of the mesentery are separated, the space becomes larger. This happens when a cyst or new growth invades the locality.

Slocum[18] states that cysts of the mesentery may be located anywhere from the duodenum to the rectum. One-half of the cysts occur in the small bowel and one-fourth in the mesentery of the ileum. The contents of the cysts may be clear, milky, sebaceous, bloody, yellow

or brown. These cysts are usually benign but malignant degeneration can occur which may be either carcinoma or sarcoma. Cysts in the region of the jejunum and upper ileum are chiefly mesenteric in distribution while in the ileocecal region they lie in the submucosa or muscular layers of the intestinal wall.

Between the two layers of the mesentery, the lymphatics drain the intestines, the efferent vessels drain the chyle to the glands and eventually bring it to the receptaculum chyli and then to the blood vessels which carry the blood to and away from the intestines.

Diversified classifications of mesenteric cysts published to date have for the most part only added to the confusion concerning the etiology and development of chylous cysts.

Thompson and Chambers[19] define cholangioma as a tumor of lymph vessels which consists of an anastomosing network of lymph spaces supported by thin walls or thick septa, is lined by endothelium and contains chyle or more inspissated fatty material which is often blood stained. Endothelium lining the cavernous lymph spaces may be flattened, or in places hyperplastic, proliferating or heaped up into layers. Some elastic tissue and bundles of smooth muscle may be present in the walls. Enteric cysts, according to Warfield,[21] have an epithelial lining of the intestinal type, whereas the cholangioma has an endothelial lining. Malignant change is rare.

Dowd,[6] Warfield,[21] Higgins and Lloyd[8] regard chylous cysts as accumulations of chyle in pre-existing cysts and not as arising from congenital abnormalities or obstruction of the lacteals or lymph channels. Other investigators, on the other hand, have found evidence that these cysts are of embryonic origin and exist as definite entities. An obstructive lymph angiectatic variety from any cause has been questioned by many because it is thought that the rich anastomosis of lymph vessels in the mesentery and the retroperitoneal region will not permit their occurrence. The embryonic origin of the lymph angiomatous variety on the other hand has many supporters.

Ewing[7] classifies mesenteric cysts as follows: (1) lymphatic or chylous; (2) enteric; (3) urogenital; (4) dermoid or teratoid cysts.

Hill[9] says all mesenteric cysts are of two types: (1) *simple*, namely, serous, chylous or irregular type; and (2) *neoplastic*, from ectoderm (dermoid), mesodermioma (lymphang), entoderm (enterocystoma), or fetal inclusion (teratoma).

Higgins and Lloyd[8] state that such adjectives as lymphatic, chylous and sanguineous have no precise bearing on the problem of etiology in the classification of cysts of the abdomen.

Beahrs et al.[2] write that chylous cysts should be classified not according to contents, location or size, but as to the histopathologic character of the cyst wall, known history and etiology. The adjectives serous, sanguineous and chylous could be used for subgroups.

Beahrs et al.[3] suggest the following classification of abdominal cysts:

A. Embryonic and developmental cysts: Examples: enteric, urogenital, lymphoid and dermoid
B. Traumatic or acquired cysts: In this group, cysts may develop following forceful injury, which in turn causes hemorrhages resulting in sanguineous cysts or rupture of lacteals with extravasation of chyle into the surrounding tissue.
C. Neoplastic cysts:
    1. Benign
        a. Hyperplasia lymph vessels
        b. Lymphangioma
    2. Malignant
        Sarcomas
D. Infective of degenerative cysts:
    1. Mycotic
    2. Parasitic (echinococcus)
    3. Tuberculosis

The symptoms of mesenteric cysts are not pathognomonic and a diagnosis of cyst is seldom made before the operation. It is seldom possible to distinguish types of cysts except on pathologic examination. The presenting symptoms are most frequently those of an acute surgical complication. They usually resemble those of intestinal obstruction as a result of volvulus, angulation of bowel, adhesions, intussusception, stenosis of the bowel or pressure. They may resemble those of peritonitis, hemorrhage, rupture of a cyst, torsion or incarceration of a cyst or of pressure on various organs. In uncomplicated cysts, a centrally located palpable tumor which usually is markedly mobile in the transverse direction may be found on examination. Abdominal distention may be noted by the patient. Pain may be generalized,

localized or associated with point tenderness. Malaise, anorexia, nausea, vomiting, fatigue and loss of weight and strength are frequent complaints. Physical examination reveals a palpable tumor.

Hinkle[10] stresses roentgenologic examination as a valuable aid for more frequent diagnosis of mesenteric cysts, based on visualization and localization of these lesions. He concluded that the cysts may be seen because of increased or decreased density, opaque inclusions or calcification within the cyst wall.

Prognosis depends on the location and size of the cyst, sites of attachment, time of diagnosis and operation, presence of complications, whether benign or malignant type of operative procedure used and the age and general condition of the patient rather than any one factor alone.

True mesenteric (lymphatic or chylous) cysts and enteric cysts (duplications) are often confused. The two lesions have somewhat similar position in the mesentery but are pathologically different and thus require different treatment.

The enterocystomas are due to cystic dilatation of a congenital duplication of the bowel. The duplication is a thick-walled structure which has all the intestinal layers in its wall. It lies immediately adjacent to the bowel between the folds of the mesentery. The blood supply of the duplication is the same as that of the adjacent intestines so that the duplication cannot be excised without injuring the bowel or its blood supply.

The chylous cyst, on the other hand, is thin-walled and has no muscular coat or mucosal lining, while it lies in the mesentery space between the folds. It can be removed without injuring the blood supply of the bowels.

Intestinal obstruction, either acute or chronic, is the most important and frequent complication of mesenteric cysts. Other complications are: (1) hemorrhage into the cyst; (2) torsion of the cyst and (3) rupture of the cyst.

Complications of chylous cysts are: (1) bowel obstruction (30–50 per cent); (2) peritonitis secondary to obstruction or rupture of the cyst, spontaneous or by direct trauma; and (3) torsion of the mesenteric cyst (rare).

A surgeon has a choice of the following operative procedures: (1) aspiration (only preliminary procedure); (2) enucleation; (3) resection and (4) marsupialization.

Roller[16] believes that enucleation carries only a 9 per cent mortality while resection mortality may reach 30 per cent.

Marsupialization carries the disadvantage of long morbidity with always the possibility of a persistent sinus, non-obliteration of the cavity and a high incidence of intestinal obstruction necessitating further surgery.

Alesen[1] is of the opinion that the treatment of choice for mesenteric chylous cysts consists in their removal by that technic which is best adapted to the case seen through the open abdomen.

Marsupialization as a type of treatment was considered obsolete by Parsons[14] in 1936 while Beahrs and Judd[2] used it successfully according to case reports in 1950.

CONCLUSION

The incidence, etiology, symptomatology and treatment of chylangioma or chylous cysts has been reviewed.

Two cases of chylous cysts of the mesentery have been presented. One was treated by enucleation. The second was treated by bowel resection with the involved mesentery cystic tumor.

Chylous cysts are rarely diagnosed before operation since they frequently cause no symptoms until complications develop. The most consistent abdominal finding that should help one in the diagnosing of the presence of a cyst is a freely movable abdominal mass. Because of their rarity, the causes of chylous cysts are difficult to establish definitely.

Our early diagnosis and improved surgical technics, and the present availability of chemotherapy, will contribute toward a considerable improvement in the morbidity and mortality rates for the removal of chylous cysts.

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Lithopedion

WITH A REPORT OF TWO CASES PRESENTING AS RECTAL LESIONS

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ECTOPIC gestation is stated to occur approximately once in 300 pregnancies.[1-3] Among the more unusual results of such extrauterine fetation is mummification or calcification of the fetus—the lithopedion or "stone child." It has been postulated[1,3,4] that a fetus which has been retained in the uterus for a considerable period of time could, by the deposition of lime salts, be transformed into a lithopedion, particularly in instances of missed labor or missed abortion. Others,[5,6] however, believe that calcification of the fetus or the membranes, or both, occurs only with extrauterine pregnancy. Masson and Simon[5] state that three conditions are essential to the production of a lithopedion: (1) the development of a fetus to at least the third month, (2) retention of such a fetus within the abdomen and (3) the absence of infection. Thus it would be doubtful if lithopedion formation would take place within the uterus, for the occurrence of resultant infection in this organ would cause destruction of the fetal tissues.

At present there are two systems of classifying the condition of calcification, in whole or in part, of the products of conception. Kuchenmeister[7] proposed a most satisfactory grouping. In his first category is the lithokelyphos, meaning stone sheath, the condition in which the calcification is limited to the fetal membranes. The fetus, which is surrounded by the stone sheath, may be subject to some change, but the calcifying process does not implicate it. His second category is that of the lithokelyphopedion, in which both the fetus and the membranes are petrified and the amniotic fluid is absent, due possibly to leakage or absorption. The third division is the lithopedion or stone child. In this instance the fetus alone has become stony or petrified by the deposition of lime salts.

Another equally adequate and actually very similar classification has been offered by Masson and Simon.[5] They use the term lithopedion to describe the entire group. Lithokelyphos and lithokelyphopedion are retained as in Kuchenmeister's grouping, and the word "lithotecnon" is introduced to describe the fetus infiltrated with calcium salts, with no or only negligible involvement of the fetal membranes.

The incidence of lithopedion formation in eccyesis has been variously reported[5,6,8,9] in the last decade as 0.81 per cent, 1.5 to 1.8 per cent, 2.0 per cent, and 1.03 per cent. Undoubtedly a decrease even in this low incidence can be expected since the diagnosis of extrauterine gestation is now made more promptly and accurately, and early surgical intervention is universal. In 1951 Anderson, Counselor and Woolner[6] reported three cases of lithopedion or lithotecnon and stated the total number of reported cases to be 252 at that time. Four additional cases have been reported[10-13] since 1951 and two cases are presented herein.

Lithopedions may affect their hosts in various ways. They may be carried for many years as asymptomatic, benign foreign bodies, sometimes being found only at necropsy,[5,14] or they may initiate distressing, disabling and perplexing symptoms, either early or late. Immediate surgical removal is indicated upon recognition.[6]

In the two cases reported herein the patients' symptoms and the initial examinations indicated rectal lesions, the first as a case of apparent fecal impaction or obstruction due to a foreign body and the second as a draining rectal fistula.

Foreign bodies of the intestinal tract are found most frequently in the sigmoidal and rectal areas because of the anatomic configuration of these organs. These foreign bodies can be grouped according to their origin, as follows:

(1) those ingested, (2) those formed within the intestinal tract, including the fecal impaction resulting from a fecalith or coprolith, (3) those from a neighboring viscus and (4) those introduced via the rectum.[15] Symptoms depend on many factors including the size and shape of the material, the duration of its presence and the amount of damage caused. The most frequent symptoms of fecal impaction are those of obstipation, coming on rather suddenly with some intestinal distention, and rectal pain.[16] There may be a frequent desire to defecate.

An anorectal fistula is an abnormal tract or communication connecting the anorectum with some adjacent viscus or skin surface. Anorectal fistulas are almost always preceded by abscess formation in the anal region.[16] Pelvirectal abscess is not uncommon and its etiology is varied, but among the factors giving rise to this condition is the traumatism arising from the presence of a foreign body.

CASE REPORTS

CASE I. An eighty-one year old white widow was admitted to the Millard Fillmore Hospital on August 20, 1952, with the chief complaints of lower abdominal pain, slight abdominal distention and obstipation of approximately thirty-six hours' duration. There was no nausea or emesis. Very little flatus had been passed from the onset of her symptoms until the time of admission. The past history was essentially negative. She had never had any similar episodes. She had had no surgery or any serious illnesses. She had been pregnant three times and had three children. Her menses, except for the three instances of pregnancy, had always been regular and had terminated thirty-two years previously.

Physical examination revealed no abnormalities other than distention and hyper-resonance of the abdomen. Rectal examination disclosed a firm, hard mass obstructing the rectum approximately 8 cm. from the anus. Pelvic examination showed a marital atrophic introitus. The uterus was small and anteflexed. Results of laboratory studies were within normal limits. The diagnosis of fecal impaction was made, and because of the patient's age it was decided to remove this obstruction under general anesthesia.

Operation was performed the same day. The rectal mass was grasped with a sponge forceps, brought into view and carefully dissected from the bowel wall to which it was attached. A small hole observed in the anterior rectal wall was closed. The patient's postoperative course was uneventful and she was discharged on the eighth day.

The specimen removed was an irregular, yellowish gray fragment of firm, osseous tissue in which two small spicules of bone could be seen. These fragments of bone measured approximately 12 mm. in length and 1 mm. in diameter; their surface was irregular and there were numerous small projections. In one area of the specimen there were several laminated fragments of bony tissue which suggested the thoracic cage in outline. Cut section disclosed some yellowish cheesy material and small particles of bone.

Microscopic examination revealed multiple fragments of partially calcified, degenerated bone, hyaline cartilage and necrotic tissue intermingled with old blood. Along one edge portions of tubal wall with preserved epithelium were present.

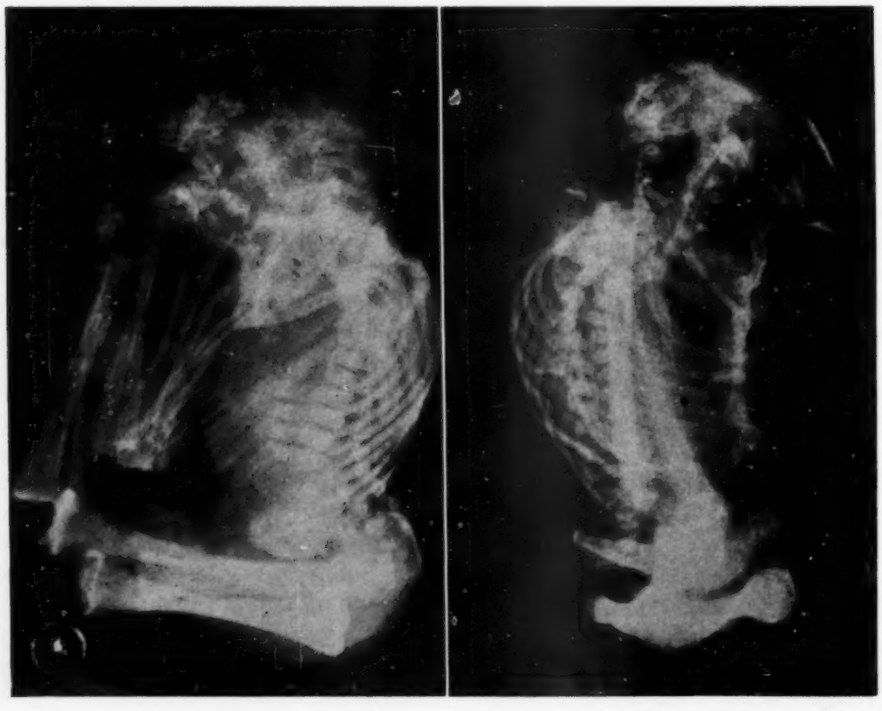
CASE II. A twenty-one year old married colored woman was admitted to the Millard Fillmore Hospital on October 29, 1953, with a history of having dull pain in the suprapubic area and in the right lower quadrant of the abdomen of about one month's duration. Following onset of this pain she said she had noticed a whitish mucoid discharge from her rectum; one week prior to hospitalization she had a rather profuse rectal discharge of a yellow, purulent fluid, after which the pain had disappeared. A whitish discharge, however, continued. Examination by her physician prior to admission revealed a marital introitus, bilateral adnexal tenderness and a fullness in the cul-de-sac. The uterus was not enlarged or remarkable in any way. Proctoscopy showed a fistula draining into the rectum.

Her past history was non-contributory and included no surgery or serious illnesses. Her menstrual periods had always been regular with the exception of two pregnancies, one at the age of fifteen and the other at the age of eighteen. Both terminated by spontaneous abortion. Subsequent to the first abortion she had had a vaginal discharge for which she received oral medication.

Physical examination showed no abnormalities except for slight protuberance of the abdomen and mild right lower quadrant tender-

1 2

FIG. 1. Lateral view of gross specimen (lithopedion) removed from abscess cavity in Case 11. FIG. 2. Contralateral view of same specimen. The formation of the rib cage, femur, tibia, fibula and other bones can be seen.



3 4

FIG. 3. In the roentgenogram of the specimen, same view as Figure 2, the bony parts can be further identified. FIG. 4. This view also clearly demonstrates calcium deposition in soft tissues.

ness. Sigmoidoscopy revealed an anorectal fistula which was surrounded by an area of inflammation and induration. Roentgenographic studies included a scout film of the abdomen, an upper gastrointestinal tract series and a mucosal relief roentgenogram. These were negative. Results of laboratory studies were within normal limits.

On November 10th a laparotomy was performed. Lysis of adhesions between the right fallopian tube and ovary and the sigmoid colon revealed an abscess cavity containing a hard calcified mass about 4½ cm. in its longest axis. This mass had eroded into the rectum. The mass was removed and a right salpingo-oophorectomy and a sigmoidal loop colostomy were performed. Three weeks later an end-to-end anastomosis of the sigmoid colon was effected. The patient's postoperative course was uneventful and she was discharged on December 12, 1953.

The specimen was a partially calcified bone structure approximately 4½ cm. in length and 2½ cm. in its widest diameter. This possessed the anatomic configuration of a fetus (Figs. 1 and 2) and consisted of multiple bones, including ribs, scapula, femur and spine. Roentgenograms of the specimen clearly defined the fetal structure. (Figs. 3 and 4.)

SUMMARY

1. The incidence and the classification of lithopedion formation are reviewed.

2. Two cases of lithotecnons or lithopedions are presented, bringing the total number of reported cases to 258.

3. In one case the patient apparently had carried her calcified fetus as an asymptomatic foreign body for over thirty years.

4. In both cases the presence of the lithopedion resulted in rectal symptoms and lesions.

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A Review of Causes of Death in 3,747 Subtotal Gastric Resections for Peptic Ulcer*

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THIS study was undertaken for two purposes: (1) to determine the mortality rate for subtotal gastric resections for peptic ulcer in the Veterans Administration Hospitals and (2) to study the pathogenesis of the causes of death so as to present suggestions for lessening this mortality.

Our statistics are based on replies to a questionnaire sent to 150 of the Veterans Administration Hospitals. The figures are not complete because data could be obtained from only eighty-eight of these. However, 3,747 subtotal gastric resections for peptic ulcer were reported. Most of these procedures were performed from 1946 to 1951; a few were done as far back as 1942.

It is interesting to note that the hospitals that furnished the data varied in size from less than 25 to 1,000 general medical and surgical beds. The smaller services were at tuberculosis or neuropsychiatric institutions. Some had Dean's Committee supervision but most did not. Visiting surgeons performed or assisted with very few of the operations. Many full-time and resident surgeons with varied surgical backgrounds participated in the series.

Sixty-five complete and unselected records of patients who died were loaned to us for review. When these charts were studied for the pathogenesis of the causes of the deaths, valuable lessons were learned that may permit reduction of the mortality rate to a small fraction of 1 per cent. The surgical death rate in this group was 2.82 per cent during the ten-year period from 1942 through 1951. It was 2.65 per cent for operations performed in 1950 and 2.51 per cent for those done in 1951. (Table 1.)

These figures parallel those of other large series. The mortality rates at the Mayo Clinic[1] for subtotal gastric resection for benign gastroduodenal disease was 1.7 per cent in 1950 (639 cases) and 2.6 per cent in 1951 (660 cases). At the Lahey Clinic[2] the rate was 2.57 per cent for 1,600 such operations as reported in 1952. Among the 893 operations surveyed at the University of Minnesota[3] from 1940 through 1949, the mortality rate was 4.9 per cent, although during the latter part of that series it fell to 2.7 per cent.

TABLE 1
MORTALITY RATES

Source	Year	Mortality (%)	No. of Cases	Comments
University of Minnesota	1940–49	4.7	893	Rate for later cases in this series was 2.7%
Veterans Administration	1942–51	2.82	3,747	
Mayo Clinic	1950	1.7	639	For benign gastroduodenal disease
	1951	2.6	660	
Veterans Administration	1950	2.65	678	
	1951	2.51	835	
Lahey Clinic	.....	2.7	1,600	For duodenal ulcer, reported in 1952

The causes of death in the sixty-five cases reviewed in detail may be classified as follows to stress the pathogenesis:

I. Deficiencies in preparation—eight patients.
   A. Inadequate nutritional preparation—five patients. These patients had lost much weight but had received inadequate preoperative blood, intravenous proteins, electrolytes and oral feedings to restore their blood volume, protein, electrolyte and nutritional balance. The deaths were due to edema of the lungs and stoma, and delayed shock.
   B. Failure to evaluate a liver deficiency

* An unselected series from eighty-eight Veterans Administration Hospitals. Approved for publication by the Chief Medical Director of the Veterans Administration. The statements and conclusions of the author do not necessarily reflect the opinion or policy of the Veterans Administration.

strongly suggested by hepatomegaly—one patient. Death occurred on the twelfth postoperative day due to liver failure.

C. Failure to take preoperative electrocardiograms in patients more than fifty years old—two patients. These died of coronary occlusion on the second and eighth postoperative days.

II. Severe hemorrhage after operations for bleeding ulcers—twelve patients.[6,8,9]

A. Inadequate or no treatment to the bleeding ulcer at operation—nine patients.

*B. Bleeding from a small esophageal erosion after a gastric resection for duodenal ulcer—one patient.

C. Insufficient preoperative blood to resuscitate the patient adequately for operation—one patient.

*D. Preoperative liver necrosis due to prolonged hemorrhagic shock[7] (two weeks)—one patient.

Of the patients who had inadequate treatment to the bleeding ulcers, one also had his hepatic artery ligated accidently, and another had severe injuries to the common bile duct. In a third, the operation was started when the blood pressure was not obtainable.

*III. Central nervous system complication—one patient (brain tumor).

IV. Pulmonary complications—eight patients.

A. Pulmonary embolism—four patients. Three were not ambulated early.

B. Atelectasis—three patients. None were treated strenuously with forced respiration, tracheal aspiration or bronchoscopy; one had aspirated vomitus; one was given massive intravenous infusions even after the diagnosis was made.

C. Pneumonia—one patient. This man had an undiagnosed septic temperature for two weeks before operation. As no adequate studies of the chest were recorded the patient may have had pneumonia at the time of the gastric resection.

V. Post-transfusion uremia—four patients. All of these patients had acid urine when transfused. No alkalinization had been attempted.[10]

VI. Operative factors—thirty-two patients.[5]

A. Peritonitis, cause undetermined—two patients. These had severely obstructed stomachs but had not been lavaged preoperatively.

B. Injury to the esophagus due to too high resection—two patients.

C. Perforation of the duodenal stump—fifteen patients: (1) Obstruction by a short antecolic jejunal loop—two patients. (2) Stenosed distal gastrojejunostomy stoma—one patient. (3) Undetermined cause (no operative report)—one patient. (4) Undetermined cause but no drain had been inserted—one patient. (5) Closure of the stump through inflamed tissue with the ulcer left in but no drainage or jejunal aspiration proximal to the stoma—seven patients.[4] (6) Removal of a bleeding duodenal ulcer without drainage—one patient. (7) Massive separation of a gastroduodenostomy—one patient. (8) Resection of 11 cm. of duodenum (inadvertent)—one patient.

D. Perforation of the transverse colon due to mid-colic artery thrombosis caused by a short antecolic jejunal loop—one patient.

E. Perforation at the gastrojejunostomy in an extremely malnourished patient with preoperative obstruction but no lavages—one patient.

F. Unrecognized rupture of the spleen at operation—one patient.

*G. Mesenteric thrombosis—one patient.

*H. Acute cardiac arrest—two patients.

I. Laceration of the jejunum by a tension suture—one patient.

J. Massive pancreatic damage with resection of suspected carcinoma—one patient.

K. Evisceration during bronchoscopy for atelectasis after aspiration of vomited blood following return from the operating room. No tension sutures had been used—one patient.

L. Gangrene of the duodenum after much trauma to it—one patient.

*M. Torsion of the jejunum with gangrene—one patient.

N. Abscess of the pancreas due to perforation of the duodenal stump into it but with no drainage into the peritoneal cavity—one patient.

O. Excessive amount of surgery (subtotal

* Presumably unavoidable deaths.

* Presumably unavoidable deaths

gastric resection after resection of gangrenous ileum in a patient with intestinal obstruction)—one patient.

CONCLUSION

1. The mortality rate in an unselected group of 3,747 subtotal gastric resections for peptic ulcer done in Veterans Administration Hospitals from 1942 to 1951, inclusive, was 2.82 per cent. It was 2.51 per cent for the operations done in 1951.

2. Detailed review of sixty-five unselected records of patients who died suggests that the deaths were probably unavoidable in seven.

3. Classification of the causes of death stressing pathogenesis is of greater warning value than mere statement of anatomic findings.

4. With observation of certain fundamental cautions the mortality rate should be a small fraction of 1 per cent.

5. These data are submitted with appreciation of the conscientious efforts of the excellent surgeons involved and with understanding of the difficulties and dangers of "long-distance" evaluation.

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Control of Hemorrhage—or Transfusion

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FROM time immemorial, bleeding has been a matter of great concern to the victim as well as to the physician. Superficial bleeding or an ordinary nosebleed in normal individuals may not be a cause of concern. However, these same simple types of hemorrhage in a patient with bleeding tendencies change the picture completely. A simple tooth extraction, a minor cut, a routine tonsillectomy or epistaxis may be followed by grave consequences and often requires heroic control measures.

Before the days of abundant transfusions and blood banks death was not uncommon in these minor accidents and procedures. Even at this late date such types of blood loss may still be fatal in remote parts of this country, to say nothing about the more remote parts of the world.

There are times when absence of the rarer blood types makes hemorrhage a real hazard. This is particularly true in hemophiliac tendencies where time is of the essence. Then again, in medical centers with our most modern facilities, at times the demand for blood in general surgery is greater than the supply. In fact the ease with which blood can be obtained often defeats the real object of the blood bank. Patients are often hastened to surgery without proper building up, even when no emergency exists. This tends to exhaust blood banks needlessly. Surgeons should begin to consider this phase more seriously so as to avoid what occasionally appears like an alarming situation.

This condition makes one wonder if it is not wise to prevent blood loss as much as possible and abolish this feeling of complacency and security induced by the existence of blood banks. Until a few years ago the tourniquet or actual ligation of bleeding vessels was the most potent means of controlling hemorrhage.

More recently vitamin K was introduced. This substance, however, is primarily of value in reducing clotting time in hypoprothrombinemia accompanying jaundice, hemorrhagic disease of the newborn and certain liver and gallbladder disease with increased prothrombin time. Vitamin K cannot be immediately effective because it has to be converted by the liver to prothrombin before results can be obtained.

Nearly fifteen years ago a preparation appeared which represented an old folklore remedy for bleeding, namely, the extract from shepherd's-purse. Other plants such as wood sorrel, rhubarb oxalis, etc., had been tried empirically for the control of bleeding. In the extract of shepherd's-purse Steinberg and Brown[20] identified the active principles as certain dicarboxylic acids (e.g., oxalic and malonic). This resulted in the clinically useful scientific preparation called koagamin.®

At first koagamin was used as a therapeutic agent for conditions causing general oozing in a wound. More recently, however, koagamin has become, in addition, an excellent prophylactic hemostatic agent especially in the rhinolaryngologic field where tonsillectomies are so frequent. The efficacy of this drug is even more striking where hemorrhagic tendencies are known to exist.

Our clinical experience with this preparation is in accord with the results reported in the literature during the past fifteen years. The following published reports covering a wide range of conditions emphasize this fact:

General surgery: thyroidectomy,[1,9] gastrointestinal,[9,21] cholecystectomy for obstructive jaundice,[9] appendectomy,[21] partial mastectomy,[21] prostatic resection,[4,14] prostatectomy.[6]

Opbtbalmology and otorbinolaryngology: Epistaxis,[3,7–9,21] pre- and post-tonsillectomies,[8,9,21] pre- and postadenoidectomies,[8,21] radical antrum operations (Caldwell-Luc), rhinophyma and other plastic surgery,[23] orbital bleeding.[21]

Obstetrics and gynecology: Uterine bleeding,[9,18,21] menorrhagia,[9] hemorrhagic fibromas,[18] postpartum hemorrhages,[18,21] pelvic enucleations,[18] placenta previa,[18] vaginal hysterectomy,[1] cholecystectomy,[1] melena neonatorum.[18]

Urology: Bleeding bladder,[21] transurethral prostatic resection,[14] hematuria,[9,12] transurethral prostatectomy.[4]

Internal medicine: Bleeding peptic ulcers,[9] hemoptysis,[17,21] hematuria of various etiologies,[9] hemophilia,[2,9,12,21] toxic thrombocytopenic purpura,[21] hemorrhagic purpura,[5,21] hemorrhage associated with jaundice,[21] leukemia[21] and chronic osteomyelitis.[21]

Although we have observed no untoward effects, it would seem wise to exercise caution in vascular diseases showing thrombotic tendencies. In our own series extending back eleven years we have had no such complication, and search of the literature reveals that no case of thrombosis has been reported with koagamin.

ADMINISTRATION

In spite of its innocuousness, practical application in its use has shown that any initial dosage above 5 cc. has proven useless and refractory. If additional quantities are indicated in continued hemorrhage or hemophiliacs, it is better to allow a time lapse of two hours or more between doses. In this way the beneficial effects from this therapy may be prolonged for days or weeks.

All statistical reports as well as our experience have shown that this drug has caused material reduction in clotting and bleeding time in nearly all cases. This reduction occurs whether clotting and bleeding times are normal or abnormal initially.

How this result is accomplished has not been explained fully, as there are still unsolved factors in the clotting mechanism of blood. Essentially the diagram devised by Howell many years ago has never been materially altered. It is believed that koagamin has a tendency to separate the calcium ions from the prothrombin, thus permitting the latter to act more quickly. (Fig. 1.)

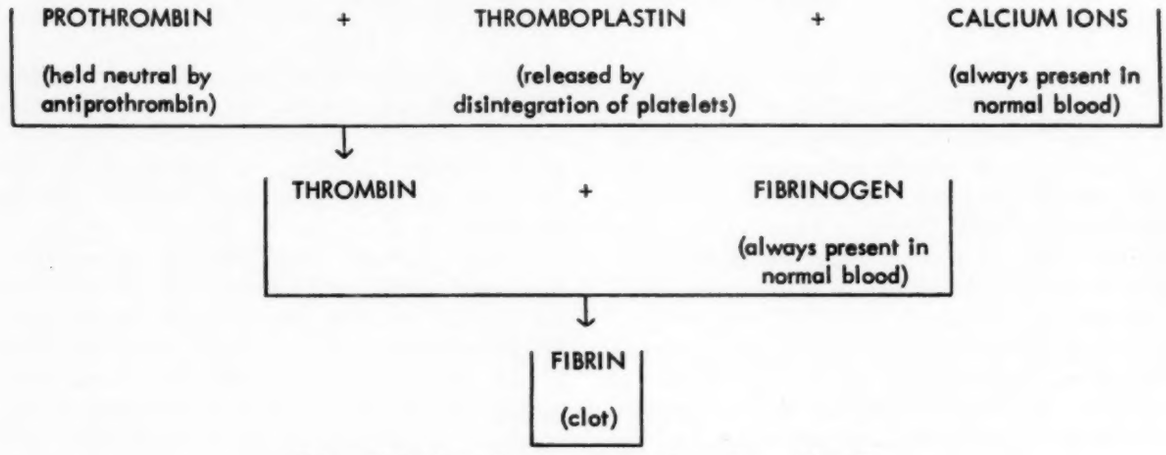

FIG. 1. Clotting mechanism of blood according to Howell.

CLINICAL MATERIAL AND RESULTS

At the Passaic General Hospital the following data were obtained from case records of the past eleven years where koagamin was administered: 542 cases of tonsillectomy and adenoidectomy;* twenty-five cases of general surgery (including twenty-three cases of pelvic, or gallbladder and bile duct surgery).

Recently a check series of twelve cases, mostly general surgery, was done against twelve control cases in which koagamin was not used. (Tables I and II.)

In this series it will be noted that in the control patients bleeding time was increased an average of 41 per cent and coagulation time was increased an average of 16 per cent one hour after operation. However, in those patients receiving koagamin the bleeding time was reduced an average of 13 per cent and the coagulation time was decreased an average of 23 per cent one hour after operation.

From our figures it is clear that in evaluating the total effect of koagamin we must also consider the increase in bleeding and clotting time caused by operation alone. We strongly suspect

* These were uniformly satisfactory from the standpoint of minimal bleeding. (About one case a year presented a problem of abnormal oozing.) Series of Dr. L. M. Matthews.

TABLE I

EFFECT OF KOAGAMIN IN TWELVE SURGICAL CASES

Case and Date Admitted	Operation	Bleeding Time		Coagulation Time	
		Before Operation	One Hour after Operation	Before Operation	One Hour after Operation
E. C., 10/17/53	Cholecystectomy	1′15″	0′30″	3′15″	3′00″
E. B., 10/21/53	Hysterectomy	1′00″	0′30″	6′00″	3′10″
C. L., 10/22/53	Hernia	1′00″	0′45″	5′10″	3′00″
A. L., 10/24/53	Bilateral hernia	1′00″	0′45″	3′05″	2′30″
R. B., 11/19/53	Postoperative adhesions	1′00″	1′15″	5′15″	5′35″
M. R., 11/17/53	Incision of foot drainage	0′30″	0′45″	5′10″	3′00″
M. T., 11/17/53	Hysterectomy	0′45″	1′00″	3′30″	2′45″
B. W., 11/19/53	Hernia	0′45″	0′30″	3′30″	3′00″
T. D., 11/24/53	Hernia	0′30″	0′30″	3′10″	3′00″
E. F., 11/24/53	Cholecystectomy	0′45″	0′45″	4′45″	4′15″
I. N., 12/1/53	Fistula of anus, hemorrhoidectomy	0′30″	0′30″	4′45″	3′00″
J. R., 12/1/53	Orchiopexy	0′30″	0′30″	3′15″	2′45″
		13% average decrease		23% average decrease	

TABLE II

STUDIES IN TWELVE SURGICAL CONTROLS*

Case and Date Admitted	Operation	Bleeding Time		Coagulation Time	
		Before Operation	One Hour after Operation	Before Operation	One Hour after Operation
G. B., 11/2/53	Saphenous ligation	1′00″	1′15″	5′00″	5′00″
A. W., 11/2/53	Cyst of breast	0′30″	0′30″	5′00″	5′00″
D. B., 11/13/53	Diagnostic dilatation and curettage	1′30″	1′30″	4′00″	4′00″
M. N., 11/13/53	Hysterectomy	0′30″	1′00″	4′30″	4′45″
A. S., 11/13/53	Hernia	0′45″	1′30″	3′30″	5′30″
B. C., 11/17/53	Ganglion of wrist	0′30″	1′00″	3′10″	4′45″
E. T., 11/19/53	Dilatation and curettage	0′45″	1′00″	3′15″	4′30″
C. L., 11/19/53	Abdominal tumor and ovarian cyst	0′30″	0′30″	5′00″	5′10″
D. C., 11/19/53	Tumor of thigh	1′30″	1′45″	4′00″	5′00″
W. N., 11/19/53	Hernia	0′30″	1′00″	4′00″	4′35″
N. M., 11/24/53	Cholecystectomy	1′00″	1′30″	3′00″	3′00″
B. S., 12/2/53	Pilonidal cyst	0′45″	1′15″	3′30″	4′45″
		41% average increase		16% average increase	

* Did not receive koagamin.

that inhalant anesthetics may play a role in this increase.

If this method is followed, the average reduced percentages caused by koagamin are: bleeding time, 54 per cent; clotting time, 39 per cent.

CONCLUSIONS

In view of the extensive work done by others and our own series of cases as summarized herein, representing the prophylactic as well as the therapeutic value of koagamin,* I believe that the following conclusions are justified:

1. Koagamin has a unique blood-clotting faculty, acting promptly and usually requiring only one or two injections.

2. It is fully compatible with vitamin K where it is indicated.

3. If given promptly and judiciously, it will often obviate the use of transfusion and avoid waste of precious blood so often required.

4. Its prophylactic use preoperatively tends to reduce blood loss and to facilitate surgical procedures.

5. Where general oozing occurs as in tonsillectomy, pelvic surgery, breast amputation and prostatic surgery, it is particularly valuable.

6. Over an eleven-year period no untoward effects have been observed with the use of koagamin in 567 cases.

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SURGICAL REVIEW

Venous Thrombosis and Thromboembolism

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THROMBUS formation in the deep veins of the lower extremity is a serious complication of any illness and one that is a threat to the life of the patient. The ideal in the treatment of this complication is prevention. However, if prophylaxis fails, the early recognition of the presence of a deep venous thrombosis and the initiation of immediate and adequate treatment is of primary importance. In this presentation a method of management which has proved satisfactory is described.

CLASSIFICATION

When a thrombus forms in the deep veins of the lower extremities, it is classified generally into two forms: phlebothrombosis and thrombophlebitis, a terminology suggested by Ochsner and De Bakey.[12,13]

Phlebothrombosis, or "bland" thrombus (Homans[9]), refers to a thrombus that is either non-adherent or very loosely attached to the intima and is without any associated inflammatory reaction. Accordingly, thromboembolism is always imminent.

The term thrombophlebitis is used to indicate the presence of an inflammatory reaction in association with the thrombosis. In this type the thrombus is of the adherent variety and the danger of thromboembolism is remote.

The concept that phlebothrombosis and thrombophlebitis are different and distinct is believed open to question. It is the opinion of the author that the pathogenesis for each is the same, but the difference in the clinical manifestations is dependent upon the rate of formation of the thrombus and its extent rather than upon the presence or absence of an associated inflammatory reaction. In thrombophlebitis it is believed that the thrombus is extensive and the rate of its formation and propagation is rapid. Conversely, in phlebothrombosis the extent of the thrombus is limited and the rapidity of its formation and propagation is retarded. This concept is based on repeated observations of patients in whom the first indication of the presence of a deep venous thrombosis was the occurrence of either a fatal or a non-fatal pulmonary embolus. In many of the patients in whom the embolus was not fatal, the first indication of its site of origin was the occurrence of pain and swelling in one of the lower extremities seven to ten days later. The end result in the affected extremity was not unlike that present in the patients with thrombophlebitis. However, occasionally in some patients in whom a pulmonary embolus is the primary manifestation of a peripheral deep venous thrombosis, recurrent pulmonary emboli may occur over a period of weeks or even months, despite which signs and symptoms related to the lower extremities may be either minimal or absent.

ETIOLOGY

The exact etiologic factor or factors in the formation of a venous thrombosis in the deep veins of the legs is unknown. Consequently, many postulates have been proposed, a discussion of which is beyond the scope of this presentation. However, there are certain predisposing causes to the formation of a deep venous thrombus that may be enumerated. These are: (1) operation, (2) parturition, (3) trauma and (4) prolonged confinement in bed, particularly in the treatment of patients with heart disease. In addition there are basic contributory factors that are commonly associated with the predisposing causes, namely, (1) circulatory stasis, (2) hemorrhage, (3) shock, (4) infection, (5) malnutrition, (6) anemia and (7) dehydration. Contrariwise, there are patients in whom venous thrombosis occurs without

any evident predisposing or contributory cause. Such occurrences serve only to emphasize our lack of knowledge relative to specific etiologic factors.

Stasis in the peripheral venous circulation is considered one of the most, if not the most, important factors in the formation of a thrombus within a vein. The return of the venous circulation to the right side of the heart is dependent upon five factors, namely, (1) the vis a tergo or the force of the heart beat; (2) contraction of the muscles in the extremities; (3) aspiration action of the thorax; (4) viscosity of the blood; and (5) the effect of gravity. Disturbances in one or more of these factors that may induce stasis in the venous circulation and predispose to the formation of a deep venous thrombus are commonly present in heart disease, malnutrition, hemorrhage, shock, trauma, dehydration, the improper positioning of the patient in bed and, finally, the use of constricting dressings.

When one considers these factors, it is difficult to explain why, in patients with paraplegia of the lower extremities and infected decubitus ulcers, deep venous thrombosis and pulmonary embolism are comparatively infrequent complications.

SYMPTOMS

The symptoms that are related to the presence of a deep venous thrombus in one of the extremities vary from patient to patient. However, when one analyzes carefully the case histories, the patients may be divided into four group patterns or categories, according to the symptoms that are manifest.

Group I. Objective symptoms present; subjective symptoms minimal or absent: The patients in this group have slight or no subjective symptoms. However, on examination of the extremities the following may be noted: (1) tenderness along the plantar surface of the feet; (2) pain in the calf on dorsiflexion of the foot (Homans' sign); (3) tenderness on compression of the calf, and on palpation a feeling of fullness or "heaviness" of the calf; (4) duskiness of the toes and distention of the superficial veins (satellite veins of Pratt) on dependency; (5) swelling of the ankle or leg. The diagnosis in patients in this group is dependent upon daily and often repeated examination of the lower extremities. Pulmonary embolism, although not an initial manifestation, may occur as a complication.

Group II. Subjective and objective symptoms present: In the patients in this group the initial complaint is pain in the calf. Upon examination of the affected extremity the objective symptoms may include one or more of those previously described. In this group, similarly as in Group I, pulmonary embolism may occur as a complication of the venous thrombosis.

Group III. Primary symptoms, pulmonary in origin; subjective and objective symptoms relative to the extremities, slight to moderate, or absent: Usually the initial symptom in patients in this group is chest pain. Dyspnea, cough and hemoptysis, either singly or combined, may be present. All too frequently the primary diagnosis is pneumonia, particularly virus pneumonia. Concomitant with the pulmonary symptoms, objective evidence of a venous thrombosis may be present. However, these objective symptoms may not occur for seven to ten days. They even may be minimal or persistently absent.

Group IV. Acute thrombophlebitis and acute fulminating thrombophlebitis (phlegmasia cerulea dolens): In patients who comprise this group the venous thrombus is extensive and forms rapidly. It is frequently called the adherent or inflammatory type of thrombosis, relative to which the complication of thromboembolism is considered less likely to occur. Depending upon the character and the severity of the symptoms, there are two clinical types of thrombophlebitis.

Type I: Acute thrombophlebitis. This is the type most commonly observed. It is characterized usually by moderately severe pain in the affected extremity, (commonly the left), fever (102° to 104°F.) and frequently chills. On examination of the extremity diffuse swelling, "heaviness," tenderness, dusky hyperemia and an increase in the skin temperature are noted. A diminution in the femoral arterial pulsation is generally observed.

Type II: Acute fulminating thrombophlebitis (phlegmasia cerulea dolens). In this type the local and systemic reactions of the patient to the disease process are more acute and more severe. Shock is frequently present and death may occur within thirty-six to forty-eight hours. Chills are frequent and the fever is high (104° to 106°F.). The involved extremity is massively swollen and tender. The swelling

may extend to the area of the sacrum, the lower portion of the abdomen and to the opposite extremity. Cyanotic rubor with diffuse petechial hemorrhages is commonly present. The massive swelling of the extremity and the associated reflex arterial spasm may be sufficient to occlude completely the peripheral arterial pulsation and simulate an embolic occlusion (pseudoembolic thrombophlebitis). In fact, ischemic necrosis of a varying degree may occur and require minor or major amputation of a part. Furthermore, even though the "local" reaction associated with the venous thrombosis is severe, pulmonary thromboembolism may and does occur. Indeed, in some patients such a complication has been the immediate cause of death.

DIAGNOSIS

The diagnosis of deep venous thrombosis in one of the extremities is frequently a simple one, but in some patients it may prove most difficult. However, if one considers the possibility of its presence, a careful examination of the extremities daily, and even twice daily, may aid in the making of an early diagnosis.

Allen[1] has emphasized repeatedly the significance of a concomitant rise in temperature, pulse and respiration as an early manifestation of an occult venous thrombosis. In fact, when such occurs prophylactic ligation of the superficial femoral vein bilaterally is advised.[1] Pain and tenderness involving the plantar surface of the foot, pain referred to the calf on dorsiflexion of the foot (Homans' sign), dusky rubor of the toes and distention of the superficial veins (sentinel veins of Pratt) on dependency are all indicative of deep venous thrombosis in an extremity. In addition, palpation of the calf will frequently demonstrate tenderness and a concomitant "heaviness" in the tissues, with diminution in the normal flabby consistency and a local increase in the skin temperature.

The occurrence of pulmonary infarction postoperatively is usually prima-facie evidence of a non-adherent or loosely adherent venous thrombus. Unfortunately and all too frequently, this primary manifestation of deep venous thrombosis may be the immediate cause of the patient's death. In patients with mitral valvular disease, congestive failure or both, in whom a pulmonary infarct occurs, one should consider always the possibility of a stasis infarction within the pulmonary circulation rather than a thromboembolism originating in a thrombosis of one of the deep veins of the lower extremities.

A thrombus in the superficial or saphenous vein, particularly if it is not a varicose vein, may be indicative of primary thrombosis in the deep veins. It is believed that in those instances in which thromboembolism is associated with venous thrombosis in the superficial system, the true origin of the embolus is most commonly an unrecognized deep venous thrombosis in the same or in the opposite extremity.

TREATMENT

In the treatment of patients in whom deep venous thrombosis in one of the extremities is present, rigid individualization is practiced. However, there are certain basic principles of prophylaxis which are believed important and generally applicable. Accordingly, a discussion of both the prophylactic and definitive methods in treatment is presented.

Prophylactic Treatment

Prophylactic measures may be used in the preoperative preparation, during the operation and in the postoperative care of the surgical patient.

Preoperative Preparation. Every patient upon whom an elective operation is planned should be screened carefully. Dehydration and increased viscosity of the blood, predisposing factors to venous thrombosis, should be corrected by adequate fluid and electrolyte replacement. Similarly, existing anemia or nutritional and metabolic disturbances should be treated adequately so that the patient is in the best condition possible for the operation that is contemplated. If varicose veins are present, elastic bandage supports should be prescribed or a preliminary corrective operation performed, depending upon the exigencies of the individual case.

Safeguards during the Operation. It is firmly believed that the incidence of deep venous thrombosis as a postoperative complication will be lessened if undue trauma to tissues is avoided. This may be accomplished by strict adherence to the tenets of Halsted. These tenets embody: (1) absolute hemostasis, (2) gentleness and care in the handling of tissues, (3) the use of fine sutures and ligatures,

preferably silk, and (4) the avoidance of mass ligatures and the unnecessary strangulation of tissues. In any operation, and particularly in those that are prolonged and require radical resection of large areas of tissue, shock may occur. The prevention of shock by the maintenance of an adequate fluid and blood volume is essential. This is best accomplished by the continuous and adequate replacement of blood loss by transfusions of whole blood, either intravenously, intra-arterially or both.

Postoperative Care of the Patient. The prevention of circulatory stasis is believed to be one of the most important factors in the prophylaxis of venous thrombosis. Although it is stressed relative to the postoperative care of the patient, it is just as important in the preoperative period of preparation and during the operation. If stasis of the circulation is to be prevented one must: (1) maintain an adequate circulating blood volume; (2) provide for an uninhibited respiratory exchange and adequate pulmonary ventilation; (3) induce the patient to exercise the leg muscles actively soon after operation; (4) change the position of the patient frequently; (5) avoid flexed positions of the knee and hip joints and dressings that constrict; (6) use the Trendelenburg position where indicated as an aid in the return flow of blood to the heart. Although early ambulation may not lower the incidence of deep venous thrombosis, it should be practiced provided it does not prove to be an added strain and inconvenience to the patient. The use of anticoagulants and/or ligation of the superficial femoral vein bilaterally as a prophylaxis for deep venous thrombosis is neither *practiced* nor recommended.

Definitive Treatment

In the discussion of symptoms the patients were classified into four groups according to the clinical manifestations that were present. In discussing treatment the same group classification is used.

Group I. Objective symptoms present; subjective symptoms minimal or absent: The treatment of patients in this group is conservative. Initially, absolute bed rest is prescribed and the affected extremity is elevated on a pillow. The anticoagulant, heparin, is administered intermittently by the intravenous route. When used in this manner daily determinations of the bleeding and coagulation times are not required. The anticoagulant is administered for nine days in a gradually decreasing dosage. The first three days the daily dosage of heparin is 150 mg. (15 cc.), 100 mg. (10 cc.) and 150 mg. (15 cc.) given at intervals of eight hours (7 A.M., 3 P.M. and 11 P.M.). The second three days the daily dosage is decreased to 100 mg. (10 cc.), 75 mg. (7.5 cc.) and 100 mg. (10 cc.). During the last three days it is further decreased to 75 mg. (7.5 cc.), 50 mg. (5 cc.) and 75 mg. (7.5 cc.). The administration of heparin in smaller doses (50 mg.) and at more frequent intervals (four hours) is preferred by many. However, the previously described method of administration has proved satisfactory.

Dicumarol is an anticoagulant that is in popular use. The absolute necessity for a completely reliable laboratory and the variation in the patient response of the prothrombin time to a specific dose of the drug are objections to its use. The low cost of dicumarol is nullified by the added expense of the daily laboratory determinations that are required. Similar objections are applicable to the newer anticoagulant, Tromexan®. However, these objections merely express the opinion of the author.

Penicillin (300,000 units) is administered intramuscularly at intervals of twelve hours. It has been a common observation that the response of the patient to the combined use of the antibiotic and the anticoagulant is greater than when the anticoagulant alone is used.

On the second day of treatment active use of the leg muscles is encouraged by prescribed exercises. The morning of the third day, and daily thereafter, an elastic bandage (A.C.E.) support is applied firmly from the base of the toes to the tibial tubercle and the patient is allowed to walk. Early ambulation prevents atrophy of the leg muscles, improves the return flow of blood, stimulates the formation of a compensatory collateral venous circulation and inhibits the propagation of a venous thrombus.

Allen[1] has championed strongly the ligation of the superficial femoral vein bilaterally as the primary treatment of choice, particularly as a prophylaxis for thromboembolism. Admittedly, a pulmonary infarct is an ever present danger. However, the disadvantages inherent in the routine practice of a superficial femoral vein ligation are believed to exceed the proclaimed advantages. Should a pulmonary embolus occur as a complication while the

patient is being treated conservatively, ligation of the inferior vena cava rather than ligation of the superficial femoral vein bilaterally is preferred.

Group II. Subjective and objective symptoms present: The treatment for the patients that comprise this group is the same as that prescribed for the patients in Group 1.

Group III. Primary symptoms, pulmonary in origin; subjective and objective symptoms relative to the extremities, slight to moderate or absent: In this group the initial symptom of the patient, and frequently the first evidence of deep venous thrombosis, is the occurrence of a pulmonary infarct. This may or may not be fatal. In other patients in this group symptoms and signs of deep venous thrombosis in one of the extremities may be present at the time the pulmonary infarction occurs. In any event, if the pulmonary infarction is non-fatal, the main goal of treatment is the prevention of recurrent thromboembolism. Accordingly, treatment of choice is ligation of the inferior vena cava regardless of the paucity or severity of the clinical manifestations in the affected extremity. In the treatment of patients in this group many prefer either anticoagulant therapy or ligation of the superficial femoral vein. If while receiving either type of treatment a recurrent non-fatal pulmonary infarction occurs, ligation of the inferior vena cava is advised.

Disadvantages in ligating the superficial femoral vein bilaterally are as follows: (1) The site of ligation may not be sufficiently high to be safely above the thrombus. (2) It is often time-consuming and frequently may be technically difficult. (3) The collateral circulation is limited and a persistent disabling swelling of the extremity may occur. (4) A thrombus formed proximal to the level of the ligation may be the origin of a fatal thromboembolism to the lungs.

Advantages in ligating the inferior vena cava are: (1) It is at a sufficiently high level above the thrombus. (2) It effects an abundant collateral circulation. (3) It interrupts the venous circulation from both lower extremities at the one operation.

Shea and Robertson[15] reported generally unsatisfactory late results in patients following ligation of the inferior vena cava. However, in our own limited experience with eighteen patients in whom ligation of the inferior vena cava was performed for single or recurrent pulmonary thromboembolism, both the early and late results have been completely satisfactory. In five of the patients the period of follow-up was between a minimum of five years and a maximum of seven years.

Group IV. Acute thrombophlebitis and acute fulminating thrombophlebitis (phlegmasia cerulea dolens):

Type 1: Acute thrombophlebitis. The treatment of patients with this type of deep venous thrombosis is conservative. This consists of: (1) sedation, (2) bed rest with elevation of the affected extremity, (3) continuous warm, moist, saline compresses to the whole of the extremity, (4) anticoagulant and antibiotic therapy as previously described and (5) paravertebral "block" of the lumbar sympathetic ganglia using a solution of procaine hydrochloride (2 per cent). The "block" is repeated at eight-hour intervals as indicated. The usual technic of injection is the deposition of 5 cc. of procaine solution (2 per cent) in the paravertebral area at the level of the spinous process of each of the first four lumbar vertebrae, a total of 20 cc. of the solution being used. However, equally satisfactory results have been obtained by the paravertebral injection of 15 or 20 cc. of the procaine solution at the level of the spinous process of the first lumbar vertebra.

Although thromboembolism is not a common complication in patients in this group, if it should occur, ligation of the inferior vena cava is the preferred treatment. An important feature of the aftercare of patients in this group, whether or not operation has been performed, is the continued use of an elastic bandage support. This bandage extends from the base of the toes to the tibial tubercle. It is applied to the affected extremity in the morning before arising and removed in the evening after retiring. The bandage is worn daily for varying periods of six to eighteen months or longer, depending upon the individual response of the patient. When the patient is able to walk without the elastic support for ten to twelve hours and have minimal or absent subjective and objective complaints, its use may be discontinued.

Type 11: Acute fulminating thrombophlebitis (phlegmasia cerulea dolens; pseudoembolic thrombophlebitis). This is caused by an extensive and rapidly propagating type of venous thrombosis. Characteristically the patients are acutely and gravely ill. Death may occur

within thirty-six to forty-eight hours. Accordingly, treatment of necessity must be early and adequate if uniformly successful results are to be obtained. Despite the severe and intensive local reaction associated with this type of venous thrombosis, thromboembolism (frequently fatal) is not uncommon.

The use of conservative measures, as previously described, in the treatment of acute fulminating thrombophlebitis has proved unsatisfactory and is interdicted. Once the diagnosis is established, the best method of treatment is immediate operative intervention. The operative approach is dependent upon the clinical findings. If the thrombus has extended into the pelvis with associated edema of the sacral area, the lower portion of the abdomen, and beginning swelling of the opposite extremity, the surgical exposure of the vena cava is advocated. Characteristically, thrombotic occlusion of the lumen of the inferior vena cava, in association with a periphlebitic inflammatory edema, is present. The inferior vena cava is carefully mobilized and encircled by two ligatures of silk (No. 2) which are not tied. An incision is made in the inferior vena cava and the clots are evacuated. These clots usually extend up to the entrance of the renal veins, and for their removal "milking" in a retrograde manner is required. This is followed by an active retrograde flow of blood. The proximal ligature is then tied. The clots in the distal portion of the vena cava are similarly removed by a "milking" maneuver of the fingers and the distal ligature is tied. A third ligature, transfixion in type, is inserted between the proximal and distal ligatures; or if preferred, the vena cava is doubly clamped and divided between the ligatures and the cut ends occluded with transfixion sutures of No. 0 silk. In the immediate and late postoperative phase of treatment the use of the elastic bandage support, as previously mentioned, is prescribed. Anticoagulant therapy is not employed postoperatively.

This method has been used in the successful treatment of two patients. In the first patient, a twenty-five year old woman, phlegmasia cerulea dolens occurred as a complication of childbirth. At operation the lumen of the inferior vena cava was occluded completely with thrombi to the level of the renal veins. The thrombi were evacuated, and following the establishment of an active retrograde flow of blood, the inferior vena cava was ligated and divided. The recovery was complete.

In the second patient, a fifty-six year old man, symptoms occurred without any evident predisposing or contributory cause. Ischemic gangrene of the left foot occurred, for which a mid-leg amputation was done. Shortly thereafter fulminating symptoms occurred in the right lower extremity and ligation of the inferior vena cava was advised. At operation the vena cava was occluded with thrombi to the level of the renal veins. The thrombi were removed until active retrograde bleeding was observed. Ligation in continuity of the inferior vena cava was performed using three ligatures of silk (No. 2). A complementary right lumbar sympathetic ganglionectomy was also performed. The improvement in the right lower extremity was immediate and pronounced. The subsequent improvement was progressive and the extremity resumed the normal in appearance. Three months postoperatively death occurred. Necropsy was obtained. The final diagnosis was carcinoma of the body of the pancreas. The right lower extremity was normal in appearance.

In patients in whom the thrombotic process is more or less limited to the lower extremity, the operative approach may be directed to the superficial femoral vein. The clots are removed and the vein is doubly clamped, divided and ligated. The use of this method of treatment has not been employed by the author. However, complete successes with its use have been reported.[2,3,14] Objections to this method of approach are: (1) the almost constant finding of extension of the thrombus into the iliac veins and frequently into the lower portion of the inferior vena cava; (2) the danger of thromboembolism during or as a complication of the operation; and (3) the frequent occurrence of the disease process, either concomitant or delayed, in the opposite extremity.

COMMENT

There is one type of deep venous thrombosis which merits further discussion, first because of its relative infrequency, secondly because of a lack of uniformity of opinion regarding its pathogenesis and treatment, and finally because of the high morbidity and mortality associated with its presence. I refer to acute fulminating thrombophlebitis or phlegmasia cerulea dolens.

Although phlegmasia cerulea dolens was described as early as 1593 by Fabricius Hildanus,[8] it was only of relatively recent date that its diagnosis was commonly made. The reports of Trémolières and Véran[17] in 1929 and Gregoire[5] in 1938 were factors in effecting a general interest in the condition. In 1949 De Bakey and Ochsner[2] added two cases to the three previously reported[6,16,18] in the American literature. One patient, treated by operation, recovered. The other, not operated upon, also recovered but suffered the loss of the large and small toes of the left foot and all the toes and part of the dorsum of the right foot. In 1950 Haimovici,[7] in a review of the world literature, collected twenty-seven cases which did not include the two cases reported by De Bakey and Ochsner.[2] In 1951 Miles[10] added one case to the fifty-nine previously reported and described the effect of ligation of the inferior vena cava. In the same year Veal[19] and his associates reported eleven cases and described the beneficial therapeutic effect of elevation and active, rapid exercise of the muscles of the extremity. Subsequent to this, Grant and Deddish[4] reported one case, Ellis and Windham[3] two cases, Ossius[14] five cases, Halligan and associates[21] two cases, Núñez Núñez et al.[22] two cases, and the author is reporting two cases herein. This makes a total of eighty-five cases of phlegmasia cerulea dolens reported up to and including April, 1953.

In a review of the protocols of many of the reported cases one is impressed by the inefficiency of all forms of conservative management. In 1948 Oaks and Hawthorne[11] were the first to report and to treat this condition by ligation and division of the inferior vena cava. The operation was performed on July 16, 1946, and complete recovery ensued. In 1950 Young and Derbyshire[20] reported a complete success after a double ligation in continuity of the inferior vena cava. The operation was performed on May 1, 1947. The preoperative diagnosis was an embolic occlusion of the left common iliac artery. However, at operation the true origin of the symptoms, namely, phlegmasia cerulea dolens, was recognized and the inferior vena cava was occluded by ligature. Recovery was complete. In 1951 Miles[10] reported a completely successful result following ligation of the inferior vena cava. At operation, performed on October 17, 1949, the vena cava was doubly ligated with silk (No. 4) and divided. To these three cases are added the two cases reported herein. The first patient was operated upon December 22, 1946, and the second on November 22, 1949. In both recovery was complete. This makes a total of five patients with phlegmasia cerulea dolens treated by ligation of the inferior vena cava, in all of whom a completely successful result was obtained.

The one common finding in the patients described, all of whom were treated by ligation of the inferior vena cava, was the thrombotic occlusion of the inferior vena cava, which occlusion most frequently extended to the entrance of the renal veins. In the case reported by Grant and Deddish,[4] operation was not performed. Necropsy showed a thrombotic occlusion of the inferior vena cava and massive pulmonary embolism. Irreversible compromise of the circulation in both lower extremities was present.

In 1949 De Bakey and Ochsner[2] reported, apparently for the first time, a complete success in the treatment of phlegmasia cerulea dolens following the opening of the superficial femoral vein, the evacuation of the contained thrombi and then the ligation and division of the vein. Ellis and Windham[3] employed the same approach in two patients, with recovery in one. In the patient who died, improvement in the appearance of the affected extremity postoperatively was observed. More recently Ossius[14] reported its use in four of five patients, with complete recovery in all.

In a review of the preceding it seems logical and justifiable to assume that the pathogenesis of phlegmasia cerulea dolens is an extensive and rapidly propagating thrombus blocking the deep veins of one or both lower extremities and most frequently associated with either partial or complete thrombotic occlusion of the lumen of the inferior vena cava. This blockage of the deep veins, in association with the accompanying extensive and severe edema of the extremity, causes a mechanical blockage to the arterial and particularly the arteriolar blood flow in the distal portion of the affected extremity. When this occurs in patients in the older age groups in whom varying degrees of peripheral arterial insufficiency already exist, ischemic gangrene of a part is a frequent occurrence. The role of secondary arterial spasm in the pathogenesis of this acute fulminating thrombophlebitis, although accepted as impor-

tant by many, is believed to be a minor one. This belief is supported by the minimal but inadequate response obtained after the use of single or multiple procaine (2 per cent) "blocks" of the paravertebral sympathetic ganglia. On the contrary, the concept of mechanical blockage is supported by the excellent results obtained, both immediate and late, after the surgical removal of the thrombi that occlude the inferior vena cava or the superficial femoral vein.

Once the diagnosis of phlegmasia cerulea dolens is established, conservative treatment is believed interdicted, despite a report to the contrary.[19] The prescribed treatment is the extraperitoneal exposure of the inferior vena cava, the evacuation of the thrombi occluding its lumen until active retrograde bleeding occurs, and then double ligation in continuity, or double ligation and division of the inferior vena cava. If preferred, phlebotomy and evacuation of the thrombi from the superficial femoral vein, followed by ligation and division of the vein, may be performed.

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Epidermoid (Epithelial) Cyst of the Phalanx

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TUMORS of the bones of the hand are comparatively rare. In a series of 1,211 bone tumors, Coley et al.[4] found a total of only twenty such cases. Of these, fourteen were in the phalanges (ten in the proximal, two in the middle and two in the distal). These lesions are uncommon and one type, the epidermoid cyst, is particularly rare and has until recently remained relatively obscure. While these cysts have a definite clinical picture and roentgenographic appearance, they are not usually suspected or accurately diagnosed, hence it seems worth while to report such a case.

The presence of epidermoid (or epithelial) cysts in the grasping surfaces of the soft tissues of the hand and fingers is a fairly common occurrence. The earliest report of such a case was supposedly by Wernher in 1855 and in subsequent accounts these cysts have accumulated quite a varied nomenclature, e.g., dermoid cyst, epidermoid cyst, implantation dermoid, epithelial cyst and so on. A distinct relationship to trauma has long been recognized and a significantly large number of cases have been described in tailors and seamstresses. However, occurrence of an identical lesion in the phalangeal bone seems to be quite rare. In fact in a recent review Carroll[3] found only nineteen such cases recorded to which he added six of his own. Since then, another such case has been reported[10] bringing the total up to twenty-six. The rarity of this lesion may be more apparent than real owing to a failure to recognize or report the condition.

CASE REPORT

R. R., a well developed white man aged thirty-nine, movie-film splicer by occupation, was admitted to the Bone Service of Memorial Hospital on October 26, 1953. He complained of swelling and pain in the terminal phalanx of the middle finger of the right hand. Past history was non-contributory except that twenty years prior to admission the patient had "smashed" the affected phalanx in a machine. The wound had healed under conservative treatment and the patient had been completely symptom-free except that the terminal phalanx had since then remained blunt and larger in size than the corresponding one on the left hand. Five weeks prior to admission he fell striking the affected digit but did not notice any particular symptoms. A week later, following relatively minor trauma while working, he noticed sudden sharp and localized pain in the phalanx followed shortly by increased swelling and exquisite tenderness. He was treated conservatively with rest without any amelioration of symptoms. However, the swelling had gradually subsided until at the time of admission, the size of the phalanx had (according to the patient) returned to the size prior to the present episode.

Local examination showed a general swelling of the terminal phalanx of the right middle finger. This phalanx was larger in all dimensions than the corresponding one on the left. The nail was deformed, being not only larger than the one on the left, but also curved in both planes. There was exquisite tenderness all over the affected phalanx especially on the palmar surface. Movements at the distal interphalangeal joint were completely free. There were no local or general evidences of infection and the epitrochlear and axillary lymph nodes were not palpable. Other physical and routine laboratory findings were all within normal limits.

Roentgenograms showed a purely lytic

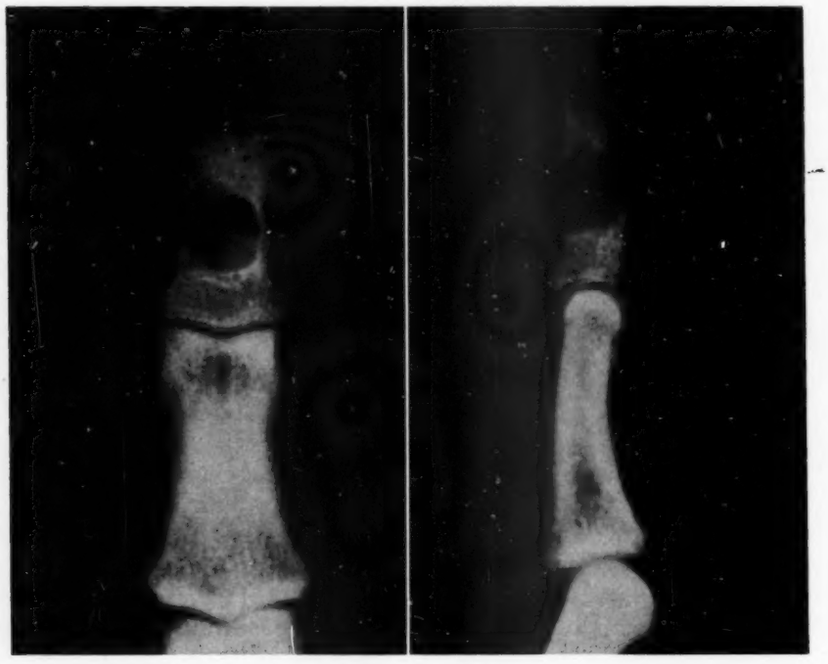

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FIG. 1. Anteroposterior roentgenogram. FIG. 2. Lateral roentgenogram; note the pathologic fracture.

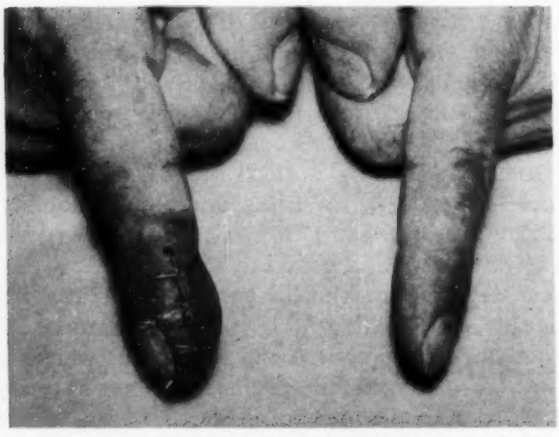

FIG. 3. Photograph of the affected finger with the contralateral normal one for comparison; note persistent bulbous swelling of terminal phalanx.

lesion occupying the middle of the phalanx and expanding the cortex on either side. A fracture line could be identified crossing the expanded cortical shell. There was no periosteal reaction. (Figs. 1 and 2.)

In view of the history, clinical findings and especially the roentgenogram, a preoperative diagnosis of traumatic epidermoid cyst (with pathologic fracture) of the terminal phalanx of the right second carpal digit was made.

On October 27, 1953, under general anesthesia, a radial lateral incision was made on the affected phalanx. The surface of the cyst was exposed and the fracture line demonstrated. The cyst wall was easily traversed and semisolid grayish white contents curetted out. Immediate microscopic examination of smears made from the contents were reported as showing keratinized debris and benign squamous epithelial cells. The cyst wall was thoroughly curetted, cauterized with a saturated solution of zinc chloride and packed with bone chips from a rib obtained from the bone bank. The wound was closed with interrupted sutures of silk. Postoperative recovery was uneventful. (Fig. 3.)

COMMENT

Possibly, because of the infrequency of the lesion, a correct preoperative diagnosis of epidermoid cyst has hitherto not been reported. Thus in 1937 Bissell and Brunschwig[2] stated that "preoperative clinical and roentgenological differentiation of these lesions from other processes such as solitary bone cyst, giant-cell tumor or chondroma, all of which may produce a unilocular cystic lesion in the bone is not possible," and again in 1953 Carroll[3] makes the statement that "where the information can be found, no correct preoperative diagnosis has been made by roentgenologist or surgeon."

In his recent review of the subject, Carroll[3] describes the usual clinical history and findings in typical cases. There is a preponderance of males, the usual age is from thirty to forty, and the majority of patients give a specific history of trauma, in spite of such traumas being rather frequent in the case of the fingers. Again, complete relief of symptoms is common following the initial trauma, swelling being noted after a variable period, about ten years on the average. Most patients seek advice not because of the swelling but because of an acute pain and tenderness in the affected finger brought about by some comparatively trivial injury which fractures an already thinned cortex. In all cases the terminal phalanx is the site of the lesion (except in one case reported by Sonntag[8] where the lesion was in the second phalanx, the third phalanx having been previously amputated), and the symptoms are sharply localized to this phalanx alone.

The roentgenographic features of this case have already been described and are apparently typical of the lesion, except that occasionally in a comparatively early case the cyst may be eccentric and there may not necessarily be a demonstrable pathologic fracture.

The gross pathologic disorder is that of a thin-walled cyst with typically white and caseous contents. Microscopy of the cyst wall in amputated specimens reveals a stratified squamous epithelium, the contents being keratinized debris.

The usual treatment has been amputation, possibly because a correct preoperative diagnosis was not made. However, in view of the nature of the lesion, a thorough curettage of the cyst with chemical cautery of the wall seems justified, at least as the primary treatment; and if there should be a recurrence, it can still be effectively treated by amputation.

There have been mainly two schools of thought with regard to the pathogenesis of the lesion. According to the first, there is a traumatic precipitation of activity in intra-osseous rests of coiled embryonal gland anlage. The more generally accepted theory is that of Gross, Reverdin and others,[8] according to whom the lesion results from the continued activity of fragmentary pieces of epidermis driven into the phalangeal bone by trauma. In fact Behrens' paper[1] has photomicrographs* showing the actual path taken by such fragments. We favor the latter theory.

* Figures 1 and 2 of the Behrens article.[1]

The diagnosis of the lesion should not be difficult provided a correlation is made between the history, physical findings and the roentgenogram.[5,6] The fact that these lesions are purely lytic and have invariably occurred in the terminal phalanges is in itself a pointer. All causes of multiple lytic lesions (e.g., osteitis fibrosa generalisata, multiple myeloma and metastatic carcinoma) can immediately be ruled out by the fact that this is a solitary lesion. The completely intra-osseous situation of the lesion makes differentiation from a pressure defect in the bone caused by an extrinsic soft part tumor, such as a xanthoma of the tendon sheath, easy. A unicameral bone cyst of the terminal phalanx is an extremely rare entity (if it exists at all) and is usually found before the age of twenty. Besides in unicameral bone cysts there is no history of trauma and the lesion is asymptomatic until there is a pathologic fracture. A giant cell tumor at this site is also unusual. The history is comparatively shorter and the roentgenograms may show fine trabeculation traversing of the lesion. The primary differentiation is thus from enchondroma, which is not only the commonest tumor at this site but also has a fairly similar radiologic appearance. However, the cystic space usually shows a characteristic radiopaque calcific stippling, the age incidence is earlier, and even if there be a history of trauma the silent interval is usually shorter. Final differentiation in such cases must thus ultimately depend upon the operative findings and microscopy.

The purely lytic nature of the lesion and the total absence of any periosteal reaction is sufficient to exclude inflammatory conditions and neoplasms like ostegenic sarcoma and Ewing's sarcoma. Moreover, these latter lesions are almost never found in the phalanges.

SUMMARY

A case of epidermoid (epithelial) cyst of the terminal phalanx of a finger is described.

The clinical and radiologic features are briefly discussed.

It is believed that in view of the definite signs and symptoms the correct diagnosis of the lesion should not be as difficult as the existing literature seems to indicate.

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Half the abdominal operations performed on patients over seventy years of age are on the biliary tract. This is not surprising since routine autopsies on patients over the age of seventy show the presence of gallstones (with or without complications) in over 50 per cent. Biliary tract surgery is now relatively safe on the aged. Mortality rate, in spite of ever possible complications, is less than 10 per cent. Advanced age, therefore, is not a contraindication to surgery. (*Richard A. Leonardo, M.D.*)

Pulmonary Infarct Simulating a Mass Pulmonary Lesion in a Patient Being Evaluated for Mitral Commissurotomy

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THE purpose of reporting this case of misdiagnosed pulmonary infarction is not because of its rarity; in our own experience and in the experience of physicians dealing routinely with pulmonary disease, this lesion is misappreciated frequently to a discouraging degree. Our aim here, especially in view of recent articles[1-4] stressing the need for prompt thoracotomy for patients exhibiting suspicious and unexplained lung lesions, particularly if they are silent, is to emphasize the fact that pulmonary infarction may simulate numerous types of chronic lung disease. Further, the need for thorough evaluation of preoperative cases, regardless of the major complaint, is brought into proper perspective by avoiding focus on a single lesion. For example, we are not evaluating a heart, rather, a patient with a heart. Obviously, this patient did have an operable cardiac lesion, but its importance was relegated to a secondary position.[3]

CASE REPORT

A thirty-nine year old white housewife was first seen in the Hahnemann Hospital Out-Patient Department in the Cardiac Clinic, referred there by her family physician for evaluation for mitral valvular commissurotomy.

Her history indicated she first knew of a heart lesion two years previously. A childhood history suggesting rheumatic infection was present only in the form of occasional "rheumatism." The entire review otherwise was insignificant. Progressive cardiac disability developed within the past two years.

In this last period, while she was under the care of her family physician, a sporadic cough developed, productive of variable quantities of sputum, often blood-streaked in the morning. At no time did she experience chest pain, cyanosis, tachypnea, acute dyspnea or "attacks" of any sort. No historical or overt evidence of paroxysmal heart action, phlebothrombosis or thrombophlebitis could be manifested.

Six months before reporting to us, signs of congestive heart failure gradually developed. Despite her decreasing vital capacity, the patient not only managed her own household (physical) chores but also augmented the family income by working full-time in a factory as a "checker" of coils in a radio-condenser assembly line, a sedentary job.

Three months after her visit to the Cardiac Clinic she was hospitalized because of noticeably diminished functional capacity despite maintenance digitalis and mercurial diuretic therapy. Her last pre-hospitalization period of three weeks was spent at bedrest.

For one year prior to her first visit to the Cardiac Clinic the patient had been taking digitalis (digitalis folia 0.1 gm.) daily, with mercurial diuretic injections (exact quantity unknown) on a biweekly basis. Interrogation from the general standpoint documented an undiagnosed febrile illness at age four, frequent sore throat in childhood and frequent upper respiratory infections throughout her life. She had four successful, full-term, normal deliveries before age thirty-six. Only with the first of these did she have any disturbance, namely, polyarthralgia of both knees.

Physical examination of this average-appearing woman with a slight degree of pallor was unremarkable in all details with the exception of the cardiovascular system. Her blood pressure was 110/58 mm.; Hg average in the supine position. Regular sinus rhythm at a rate of 86/minute was present.

FIG. 1. Lateral and postero-anterior roentgenograms showing the pulmonary infarct (I) which simulated a mass pulmonary lesion in a patient being evaluated for mitral commissurotomy.

A sharp first sound at the apex was heard. A grade II, harsh, high-pitched, systolic murmur was present, referred rather widely, particularly toward the base. An easily definable, low-pitched, rumbling mid-diastolic murmur with appreciable pre-systolic accentuation also was audible in the mitral area. The pulmonic second sound was accentuated two to three plus. Aortic area auscultation produced no abnormalities, although in the second or third interspace and along the upper left sternal border, the mitral murmurs were distinctly audible, although of diminished intensity.

Venous pressure was not increased. Breath sounds were diminished in the right lower lobe posteriorly with no associated abnormalities. The liver was enlarged 8–10 cm. below the costal margin, measured at the line extended from the right mid-clavicular.

Basic laboratory studies were well within the bounds of conventional normal standards with the possible exception of the 6 per cent eosinophilia of the peripheral blood. Serologically negative blood (Kolmer and V.D.R.L.) was Rh+, A_2 in type. Serial smears and cultures of sputum and gastric washings for tubercle bacilli all were negative.

Radiologic study of the heart showed two plus left atrial enlargement, also two plus enlargement of both the inflow and outflow tracts of the right ventricle. Left ventricular enlargement was absent. A large, comparatively homogeneous density was observed in the lower or middle lobes of the right lung. (Fig. 1.) This same lesion had been noted on chest film studies made at the time of her only outpatient visit three months prior to hospital admission. Transient first-degree A-V heart block was seen on the electrocardiogram, along with minor, non-specific T wave and S-T segment changes in multiple leads.

Intracardiac surgery was cancelled and intensive study of the pulmonary lesion begun. Serial and special projectional films were of no additional diagnostic help and the pulmonary lesion remained static.

Bronchoscopic diagnostic examination was done,[2] showing the right main bronchus stenosed to the degree that the 7 mm. bronchoscope could not pass to the middle lobe level or into the lower lobe. There was no ulceration. Mucous membrane surfaces on the right side were inflamed.

Because of these developments it was deemed wise to perform lung surgery (lobectomy or pneumonectomy) on the right, leaving mitral commissurotomy for a later date.

At surgery, there was a considerable amount of thin, brownish pleural effusion. Several adhesions joined the right lower lobe and parietal pleura, as well as the right lower and right middle lobes. There was a large gray area occupying the entire basal portion of the right

lower lobe. This area was firm, well demarcated and solid in consistency. It was triangular in shape. The superior segment of the right lower lobe was normal and well aerated. The right middle lobe was discolored slightly, presenting a grayish orange appearance on its inferior aspect. The entire right middle lobe contained no aerated tissue.

Pathologic examination of the specimen showed little other than pulmonary infarction. Details of this cytopathology are unnecessary here.

SUMMARY

1. Undiagnosed pulmonary lesions, particularly if they are silent, justify thoracotomy for diagnostic purposes.

2. Pulmonary infarction may present confusing clinical situations, even mimicking significant bronchostenotic lesions as determined by expert bronchoscopy.

3. Total-patient appraisal rather than organ-oriented, myopic evaluation is mandatory if the patient is to receive appropriate therapy.

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Intrapericardial Dermoid Cyst

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DERMOID cysts are one of the most common benign tumors found in the mediastinum. Dermoid cysts of the pericardium, however, are rare. Survey of the available literature reveals only a few scattered reports.

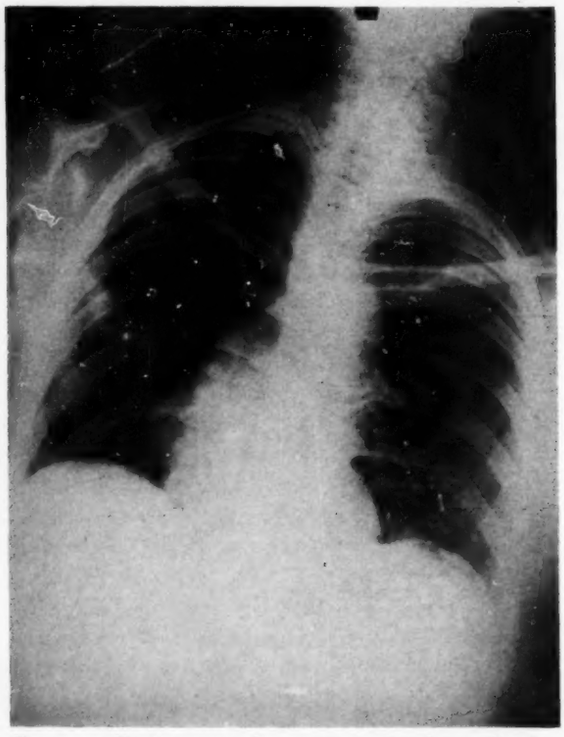

FIG. 1. Postero-anterior x-ray showing circular density in the right lower lung field.

The following case appears to be sufficiently unusual to warrant this additional report.

CASE REPORT

This fifty-seven year old white woman was admitted to the Robert Packer Hospital on September 20, 1952, with a sudden massive subarachnoid hemorrhage. Physical examination disclosed a semicomatose patient with a stiff neck, hypoactive reflexes and bloody spinal fluid. The routine laboratory examination was within normal limits.

Roentgenograms of the chest disclosed a circular density, approximately 4 cm. in diameter, in the right lower lung field, extending laterally from the cardiac border. (Fig. 1.) Left oblique and lateral roentgenograms of the chest revealed a round uniform density which could not be separated from the cardiac silhouette.

Bilateral carotid arteriogram showed a large saccular aneurysm which was apparently responsible for the subarachnoid hemorrhage. Surgical ligation of the aneurysm was performed on October 3, 1952. Postoperatively, bronchopneumonia developed, and the patient remained comatose and died on the sixty-third hospital day.

The principal autopsy findings were massive subarachnoid and intracerebral hemorrhage, extensive encephalomalacia of the right cerebral hemisphere and bilateral confluent bronchopneumonia. Examination of the thoracic contents revealed a cyst, 5 cm. in diameter, situated on the right anterolateral aspect of the pericardium. (Fig. 2.) It was smooth-surfaced, thin-walled and lay between the leaves of the parietal pericardium. There were no attachments to the lung, sternum, heart or great vessels or other thoracic structures. It did not appear to impinge on the heart muscle itself. On section the cyst contained a greasy sebaceous-like material as well as a large quantity of hair.

Section of the lesion showed a cyst wall composed of fibrous connective tissue with an outer covering of flattened mesothelial cells and an inner lining of somewhat poorly preserved stratified squamous epithelium. Remnants of hair follicles and sebaceous glands were also observed. Focal infiltrations of lymphocytes and mononuclear cells were present throughout the cyst wall. In some regions the cyst lining was fairly well preserved and focal calcification was also noted. (Fig. 3.) Diagnosis was intrapericardial dermoid cyst.

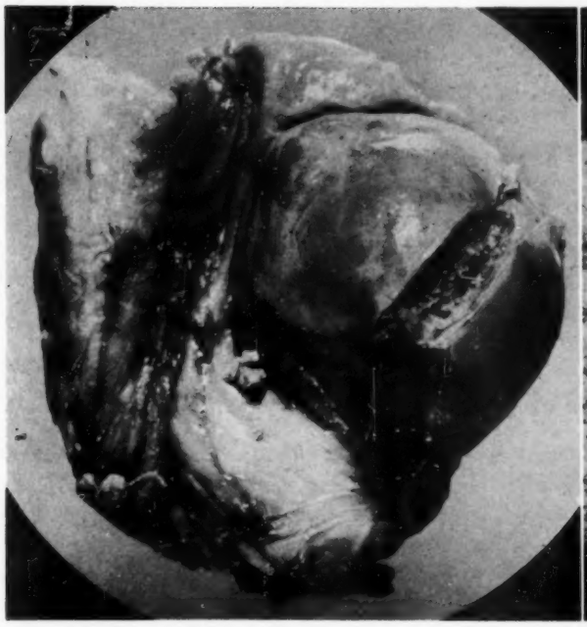

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FIG. 2. Gross specimen disclosing intrapericardial dermoid cyst.

FIG. 3. Photomicrograph showing lining of squamous epithelium and focal calcification in the wall. Hematoxylin and eosin stain × 120.

COMMENT

In the present case the intrapericardial lesion provoked considerable discussion prior to the patient's death. Initially, the possibilities given the most consideration were peripheral lung tumor, lung abscess or mediastinal cyst. On x-ray examination it may be extremely difficult to differentiate between these lesions. Occasionally the presence of a fluid level may aid in diagnosis of a cyst or abscess. The presence of bones or teeth indicate a teratoid tumor. Most frequently the true nature of this group of lesions is not revealed until exploratory thoracotomy has been performed.

By definition a dermoid cyst is congenital and derived from ectoderm and mesoderm. An epidermoid cyst contains elements derived only from ectoderm while a teratoma includes elements derived from all three germ layers. These three types of cysts comprise the majority of all mediastinal cysts. Their origin is considered to be from cell rests or from embryonic displacements of branchiogenic cells in the thorax by the descent of the heart and diaphragm.[3]

Although intracardiac teratoid tumors have been reported,[1] intrapericardial dermoid cysts are extremely rare. The majority of the pericardial cysts are of the celomic type ("spring water cysts").

Since malignancy in dermoid cysts in any location is not infrequent and since most of the mediastinal cysts eventually cause trouble, the procedure of choice is exploratory thoracotomy with surgical removal if possible.[2,4]

SUMMARY

A rare and unusual case of intrapericardial dermoid cyst is presented and discussed.

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Reduplication of the Gallbladder and Cystic Ducts

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THE recognition of reduplication anomalies of the extrahepatic biliary system occurred at least as early as ancient Hebrew times when certain variations were described in relation to the religious codes of the time. Yet as late as 1926, when Boyden[1] reviewed the reports of supernumerary abnormalities of the biliary tract, only twenty human cases had been collected. Since 1926 less than fifty additional cases have been reported. This is a report of another case of reduplication of the gallbladder and cystic ducts.

That this condition is rarely encountered in the human is pointedly shown above. The paucity of attention focused on this type of developmental anomaly in standard texts on pathology and in the literature further attests to its infrequent occurrence. Indeed, only one text reference could be found.[2] In one collected series of over 19,000 cases, including cadaver dissections and cholecystograms, only five reduplications of some form were found (an incidence of 0.02 per cent). In other mammals the incidence ranges from 12.5 per cent in domestic cats to 0.5 per cent in pigs.

With an increasing amount of surgery being carried out on the biliary system the importance of this type of pathologic condition, even though rarely encountered, should become more and more apparent, both in regard to the technical aspects of the surgery and the diagnosis of complaints referable to the biliary tract.

Boyden[1] describes five congenitally distinct types of accessory vesicles, as follows:

(1) Arising from a pancreatic anlage
Pancreatic bladders: first found by DeGraaf in 1664; found in domestic cats only

(2) Arising from a hepatobiliary anlage
Cleft gallbladders: originate by initial subdivision of the embryologic primary cystic diverticulum; chiefly characteristic in cats but present in some human cases
Diverticular bladders: vesicles or lobes arising as buds from the neck of the embryonic gallbladder; chiefly characteristic of ungulates
Ductular bladders: supernumerary vesicles derived from hepatic, cystic or common bile ducts; chiefly characteristic of humans but also present in cats; this can be further subdivided into the Y-shaped type (the two gallbladders usually adherent and occupying the same fossa with the two cystic ducts uniting before entering the common duct), and the ductular type (the two gallbladders usually separate, sometimes occupying different lobes, and with the two cystic ducts entering the common duct separately)
Trabecular bladders: vesicular outgrowths of the liver trabeculae bordering the fossa; rare; found only in cattle, sheep and possibly cats

Our case is actually a combination of the Y and ductular types of ductular bladders in that the two gallbladders were adherent and occupied the same fossa with the two cystic ducts entering the common duct separately.

CASE REPORT

E. N. (No. 34625), a forty-eight year old white woman, para IV, was first seen in a physician's office in January, 1953, complaining primarily of suprapubic pain with menses and with early menopausal symptoms. During the interview it was determined that the patient avoided fatty foods, cabbage and apples. Occasionally after dietary indiscretion the patient experienced mild right upper quadrant distress

and flatulence. The patient had had an anterior colporrhaphy and appendectomy twenty-one years before, and prior to that had had a tonsillectomy. Physical examination at this visit showed essentially: a mild essential hypertension (150/90), right upper quadrant, subcostal tenderness, and a lacerated, eroded and cystic cervix. A cholecystogram obtained on February 6, 1953, was reported as showing "multiple biliary calculi in an otherwise normally functioning gallbladder."

The patient was admitted to Butterworth Hospital on February 18th, with the history as recorded. Positive physical findings were the same. The serology was negative; urinalysis was within normal limits; red blood cell count was 4.15 million with 82 per cent hemoglobin (11.8 gm.); white blood cell count was 8,200 with 51 per cent neutrophils and 49 per cent lymphocytes. Diagnoses of (1) essential hypertension, mild; (2) chronic cystic cervicitis and (3) cholelithiasis were made. The patient was operated upon February 19, 1953.

At operation dilatation and curettage with wide conization of the cervix was first performed. Following this, through a right upper quadrant right rectus incision, exploration

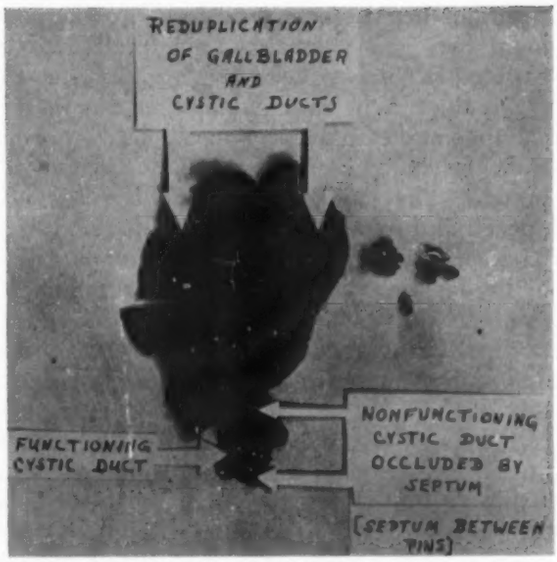

FIG. 1. Reduplication of gallbladder and cystic ducts.

showed a thin-walled gallbladder containing several small calculi. The spleen was two times the usual size and the right ovary contained a serous cyst the size of an English walnut. The remaining abdominal and pelvic viscera were normal. Retrograde cholecystectomy was then begun. The cystic artery had its origin from

FIG. 2. Cholecystogram showing double gallbladder with gallstones in one compartment.

the right hepatic artery posterior to the common duct and passed superolaterally to the gallbladder fossa, coursing anterior to the cystic duct. When the cystic duct was doubly clamped and transected, approximately 0.5 cm. from the common duct, it was seen to have two lumens. The gallbladder, after excision, was opened and found to be divided into two compartments of equal size. Each compartment had a narrow cystic duct which coursed to the common duct. The posteromedial (non-functioning) compartment contained dark, viscid bile, mucus and several small, rough-surfaced stones. The cystic duct leading from this compartment was completely occluded approximately 1.0 cm. from the common duct by a thin septum. The anterolateral (functioning) compartment contained normal bile and communicated with the common duct by a narrow cystic duct. (Fig. 1.)

The pathologic report on the gallbladder was as follows: Grossly, the gallbladder was 7 cm. in length and 4 cm. in diameter. It was completely divided into two portions of equal size. Each of these portions had a separate cystic duct. One of the cystic ducts was completely occluded by a thin septum. The mucosa of the gallbladder was smooth. Both portions of the gallbladder were opened; received separately 3 stones ranging in size from peppercorns to cherry pits. Microscopically, sections of both portions of the gallbladder showed some increase in fibrous tissue in the mucosa and the remainder of the wall, but were not remarkable otherwise. Sections of both cystic ducts showed the architecture of this organ. Diagnosis: cholelithiasis; double gallbladder.

The postoperative course was uneventful and the patient was discharged from the hospital on her sixth postoperative day. At the time of writing the patient remains free of her gastrointestinal symptoms.

The cholecystograms, when viewed with the aid of retrospection, clearly define two compartments as seen in Figure 2. The two gallbladders overlap one another to some extent. This could conceivably be confused with a phrygian cap but it should be remembered that the phrygian cap septum usually occurs more distally in the gallbladder and the cap is smaller in size than the globular shadow shown.

SUMMARY

Reduplication anomalies of the extrahepatic biliary system occur rarely. One series of 19,000 cases showed an incidence of 0.02 per cent.

Five types of such anomalies have been described. Actually, combinations of these types also occur.

A case of reduplication of the gallbladder and cystic ducts is presented.

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Perithelioma of the Ileum with Intestinal Obstruction

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PERITHELIOMA is a rare tumor, usually of low grade malignancy, which arises from perithelium, the connective tissue accompanying capillaries and smaller vessels. It arises from adventitial cells of blood vessels and forms masses of neoplastic cells surrounding blood vessels. Most pathologists do not recognize peritheliomas as distinct from sarcomas. Perithelioma produces symptoms by encroachment on adjacent structures.

Perithelioma is a rare cause of small bowel obstruction. Neoplastic growths are much rarer in the small bowel than in the colon and rectum. Hunt[2] found seven cases of neoplasm in the small bowel as compared to 385 cases of the colon and rectum, and his figures match fairly closely those of other investigators.

Faulker and Dockerty[1] in their study of small bowel tumors found that the ileum was the site of the majority of the growths, and that no malignant lymphomas involved the duodenum. They also found that lesions involving the terminal portion of the ileum tended to be a polypoid intussuscepting type, whereas those in the upper ileum and jejunum were of the ulcerative type and usually smaller than those in the terminal ileum.

Differentional Diagnosis. The differentional diagnosis of perithelioma of the ileum would include the benign tumors such as fibroma, myxoma, lipoma, neuroma, hemangioma, etc., and the malignant tumors such as carcinoma, argentaffinoma, sarcoma, lymphoma, hemangioendothelioma, hemangiosarcoma, etc. Angiomas are of congenital origin and develop from embryonic sequestrations of mesodermal tissues.

Perithelioma, endothelioma and angiosarcoma denote a vascular origin, and it is important to recognize that these several varieties of structures do not denote different types of tumor but are often found together, in varying proportions, in individual tumors.

Hemangiosarcomas are malignant proliferations of both fibroblastic connective tissue and vascular tissue. When sarcomas show a little more differentiation with numerous blood vessels and blood spaces but few or no malignant endothelial cells, they have been designated hemangiosarcomas as distinct from other sarcomas.

Sarcoma of the gastrointestinal tract is rare, with lymphosarcoma the most common form.

Hemangioma is a benign tumor composed of newly formed blood vessels and thereby differs from hypertrophy or dilatation of existing vessels.

Hemangioendotheliomas result from malignant proliferation of endothelial cells.

Ganglioneuroma is a rare tumor, usually benign, which arises from ganglion cells of the sympathetic nervous system.

TREATMENT

The treatment of small bowel tumors should consist of wide resection with adequate removal of the node-bearing mesentery, and in general, with primary anastomosis. X-ray therapy, after a wait of several weeks to prevent placing the suture line in jeopardy, is advised by many to eradicate any small foci of tumor remaining after an operation where a malignant tumor has been found.

In small bowel obstruction due to localized inflammatory process, and in the pre- and postoperative obstructive conditions, the use of the Miller-Abbott tube or a tube of a similar nature is indicated. The Miller-Abbott tube has fallen into disrepute in various areas of the country because of its difficulty in passage. However, with roentgenologic assistance the tube is generally satisfactorily passed with ease in the majority of instances.

CASE REPORT

This patient was a forty-seven year old white woman who had symptoms of epigastric pain

and vomiting starting on February 26, 1953. The pain and vomiting were intermittent. In her past history she stated she had had a hysterectomy in 1945 and a hemorrhoidectomy in January, 1953.

Examination revealed a slender individual. The abdomen was flat and non-tender, and no masses were palpable. There was a lower midline scar.

Treatment consisted of antacids, phenobarbital and belladonna. Her condition gradually became worse. Soreness and tenderness were noted in the right lower quadrant of the abdomen. She received some relief from enemas. She continued to have recurrent vomiting; tenderness also developed in the left lower quadrant of the abdomen and low grade fever developed. A barium enema revealed only diverticula to be present in the rectosigmoid area. On March 7, 1953, the patient was hospitalized. A flat x-ray film of the abdomen revealed some gas in the small intestine. The abdomen became distended, and more lower abdominal tenderness, especially in the left lower quadrant, was noted. The pains were intermittent but sharper in nature. A Miller-Abbott tube was inserted into the stomach and small bowel with the aid of the roentgenologist. The patient was then observed. Her condition did not improve and surgery was indicated.

At surgery a tumor of the small bowel was observed in the region of the lower aspect of the ileum, almost completely filling the lumen of the ileum. The entire small bowel from the upper part of the jejunum to the lower part of the ileum was greatly dilated and the serosa on the surface appeared inflamed in the lower part of the ileum. Growing into the ileum from the antimesenteric side was a firm tumor that almost completely filled the lumen of the ileum and which measured about 4 by 4 cm. in diameter. The surface of this tumor was inflamed and moderately necrotic. It had a wide base and the entire growth was markedly indurated. A few small lymph nodes were noted in the mesentery just beyond the growth. Beyond the tumor formation the small bowel was collapsed. There were no adhesions between the loops of the bowel and no adhesions between the bowel and the abdominal wall. There were moderate adhesions between the omentum and the lower anterior abdominal wall. A wide resection was made with removal of the lymph node–bearing area of the mesentery, and an end-to-end anastomosis was performed.

The pathologic report was as follows: Grossly, the specimen consisted of a resected portion of small bowel. It measured a total of 8.3 cm. in length and had been partially opened. On the opened end it measured 6 cm. in width; on the opposite end that had not yet been opened it measured 3 cm. in width. In the central portion a tumor was seen which appeared to rise from the mucosal side. It was ovoid and dome-like, and elevated from the surface approximately 3 cm. Its width was 2.5 cm. It had been sectioned through once, and on the performance of fresh sections it presented a rather homogeneous yellow-gray surface that cut with very little resistance and gave the appearance, however, of a muscle tumor. Several sections of it were taken for histologic study.

Histologic examination revealed several areas taken in several different planes. Over one or two pieces of the tumor the surface was seen overlying the mucosa. In some portions this had been exfoliated and was completely replaced by coagulated fibrinous exudate containing numerous polymorphonuclear leukocytes within its meshes. In other instances the mucosa appeared to be relatively intact. In any event, the muscularis mucosa was intact throughout with no evidence of epithelial elements transgressing it, nor was any evidence seen of epithelial tumor within the mucosa. Beneath the muscularis mucosa the stated tumor was seen which was overlaid with a considerable amount of inflammatory reaction, raising first the question as to whether or not there was neoplasm or a granulomatous reaction. In the peripheries immediately beneath the mucosa several giant cells were seen which appeared to be of the foreign body reaction type. These, however, were not persistent as one passed down into the tissue, and a pattern was seen that was quite well established and which appeared definitely to be neoplastic in type, in spite of the admixture of numerous polymorphonuclear eosinophilic leukocytes, a moderate number of lymphocytes and occasional plasma cells. The tumor had a pattern that was rather vague and loosely arranged. It seemed to be composed of a predominance of capillary and arteriolar blood vessels, large spaces between being filled with rather loosely arranged cells containing occasional inflamma-

tory cells, but being made up for the most part of fairly large cells with ovoid to round vesicular nuclei which occasionally exhibited prominent nucleoli. A moderate number of well defined mitotic divisions were seen among the cells, and on a careful study of this, the tumor cell parts. It appeared that the cells were seen in fair prominence around the arteriolar blood vessels. This had the appearance of a perithelioma. Myoblastic elements were not seen within the tumor in the sections studied. Great consideration had been given to the possibility of a neurogenic tumor, but there was no prominence of neurogenic elements here to suggest that likelihood. Neither palisading of cells nor evidences of nerve fibers were seen, and the giant cells were not nearly prominent enough to form a typical ganglioneuroma. We were thus left with the diagnosis of a perithelomatous type of tumor which appeared to be of very low grade malignancy. The fair prominence of mitotic divisions together with the considerable irregularity in size and shape of cells, the prominent nucleolar elements and the rounding up of the cellular elements in many areas suggested malignancy. From the gross examination of the specimen it appeared that an adequate excision of the lesion had been made. A careful search was made for a metastasis within the lymph node but none was found. The diagnosis was perithelioma of the small bowel with low grade malignancy.

The postoperative course was very uneventful. Penicillin, dihydrostreptomycin and intravenous glucose with electrolytes were given. The suction in the Miller-Abbott tube was continued for four days, after which the patient tolerated the diet with a minimum of gas and abdominal distress.

The patient has continued to gain weight, has no abdominal distress, and repeated periodic rechecks reveal no evidence of any recurrence of growth to date. The patient is being re-examined now at three-month intervals.

SUMMARY

A case of small bowel obstruction from perithelioma is presented. The term perithelioma is used here as a matter of expediency, even though many pathologists do not recognize this form of tumor as distinct from sarcoma. This form of tumor is unusual and is a very rare cause of small bowel obstruction. Diagnosis of the tumor can be made only by extensive histologic studies. This tumor is usually benign and causes symptoms by encroachment on and into adjacent tissues.

The treatment of this tumor is surgical, with wide resection of the bowel including the node-bearing areas of the adjacent mesentery.

The problem of postoperative x-ray in these cases is debatable. In consultation with the pathologist and roentgenologist in this case it was deemed inadvisable to give radiation therapy unless evidence of recurrence in fairly close follow-up is noted.

It is believed that the use of the Miller-Abbott tube is definitely indicated in this type of case, both pre- and postoperatively. The procedure for passing the tube should be a function of the roentgenologic department.

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Bilateral Sliding Hernia

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SLIDING hernia presents unique problems in diagnosis and repair, and is encountered just frequently enough to warrant periodic review. Williams[1] found a reported incidence of sliding hernia in from 1 to 3 per cent of all patients with hernia. Of his own patients, he found that 1.5 per cent had a sliding hernia.

However, the true incidence is difficult to determine, for the older the age group reviewed the higher the proportion of patients with sliding hernia. In slightly more than 1,000 patients with hernia upon whom I have operated, there were two with sliding hernia (an incidence of 0.2 per cent). Many of this group were of military age, in whom sliding hernia is rarely encountered.

A sliding hernia should be suspected when there is a large defect in the internal ring in a patient in whom the hernia is difficult to reduce completely and almost impossible to retain by the external pressure of a truss. Many times it may be impossible to make the correct diagnosis of sliding hernia until the sac is exposed and frequently until it is opened, unless röentgen ray examination of the colon has been made. The vital factor is to recognize it when it is encountered and neither damage the colon nor compromise its blood supply.

A sliding hernia is more frequently unilateral, but bilateral sliding hernia is encountered. In a patient who has already had a sliding hernia on one side, the second one may be suspected of being a sliding hernia, as in the case reported herein.

CASE REPORT

C. H. A., an eighty-four year old white man, was first seen in 1940 complaining of severe gnawing epigastric distress, weakness and weight loss. On examination an ulcer of the duodenum was found and considered to be responsible for his pain and anemia. An orange-sized right inguinal hernia was found to contain the cecum on barium enema. The duodenal ulcer was controlled by diet and the patient, then seventy-one years of age, did not wish to have his hernia repaired.

By the next year the hernia had become larger, could not be reduced completely and could not be retained by any one of the many different trusses which were tried. Under continuous spinal anesthesia the hernia was repaired using La Roque's maneuver (Fig. 1) to reduce the contents of the hernial sac which included the entire cecum. The right testicle and a surrounding hydrocele were excised and the hernia repair reinforced by strips of fascia

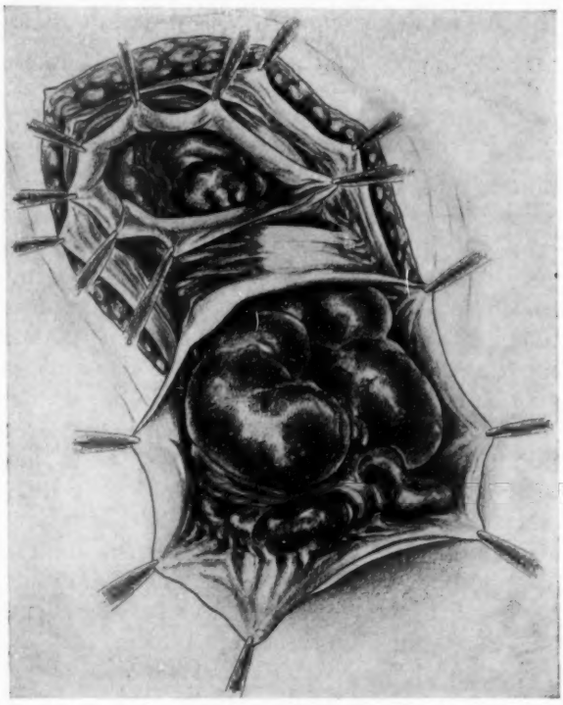

FIG. 1.* The La Roque maneuver. As soon as the hernia is identified as a sliding hernia, either because the posterior wall of the cecum or sigmoid is encountered extraperitoneally or because the intraperitoneal opening extends through only a part of the hernia, a muscle-splitting incision is made into the peritoneal cavity just proximal to the internal ring. The contents of the hernia are retracted into the abdomen through this muscle-splitting incision.

* Figures 1, 2 and 3 from WILLIAMS, C. *Ann. Surg.*, 126: 612–622, 1947.

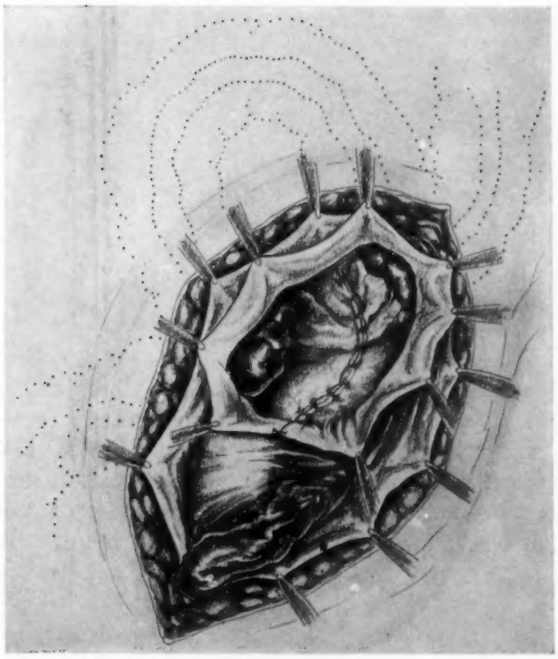

FIG. 2. The excess peritoneum has been excised and the cut edges sutured behind the sigmoid to convert it into an ordinary indirect hernia.

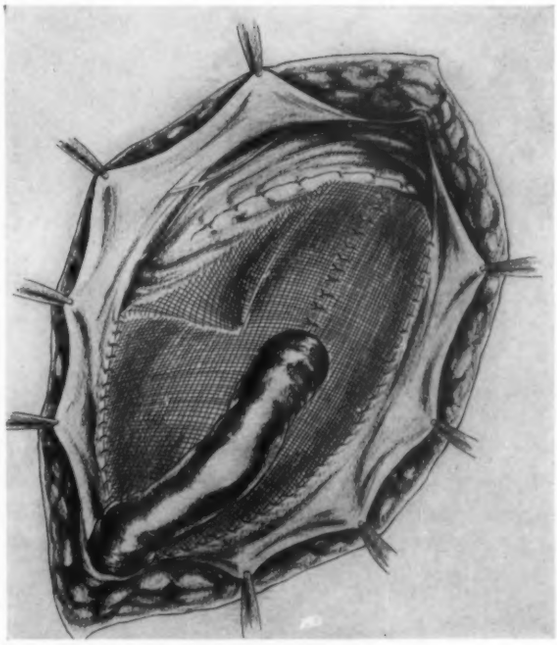

FIG. 3. The remaining defect in the peritoneum has been closed and a Halstead repair performed. The cord structures have been transplanted subcutaneously and tantalum mesh has been used to reinforce the approximation of the conjoined tendon to Poupart's ligament. It has been extended proximally to cover the muscle-splitting incision used in the reduction of the contents of the hernia sac.

lata removed from the thigh. Convalescence was rapid and uneventful.

A small asymptomatic hernia was noted on the left side one year later. It gradually became larger and was associated with severe disability. It was impossible to retain the hernia by external pressure. When the patient walked, he insisted that the pull from his hernia made him nauseated and weak. The patient's cardiovascular reserve was so poor, however, that operation was deferred until he insisted upon definitive treatment saying life was intolerable so long as he continued to have his hernia.

On the present admission, following preoperative preparation in the hospital, the left hernia was repaired under local anesthesia and the repair reinforced with tantalum® mesh. (Figs. 2 and 3.) It was a true sliding hernia. Its lateral wall was the mesentery of the sigmoid, about 12 cm. of which were incarcerated in the hernia.

Convalescence was complicated by swelling of the left testicle which did not delay ambulation or dismissal from the hospital on the sixth postoperative day. The hernia repair has remained firm and the patient's cardiovascular status has improved.

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A Clamp for Gastric Resection

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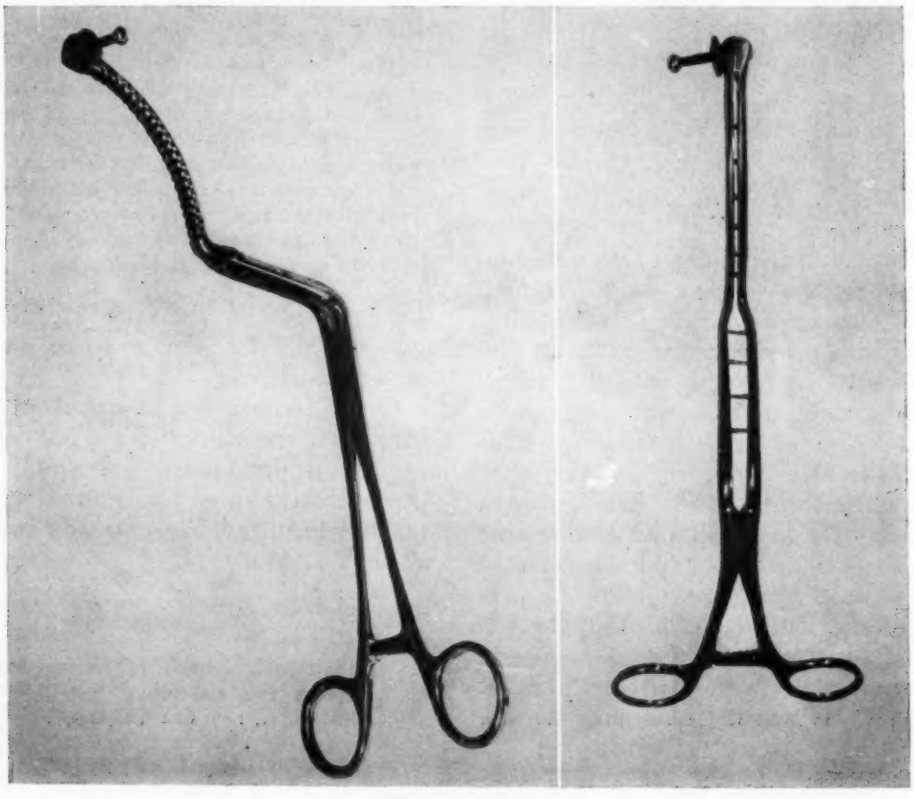
THE use of the herein described clamp has greatly reduced the difficulties sometimes encountered during the performance of a subtotal gastrectomy. This has been particularly so if the resection has been one for a high gastric ulcer, or a physiologically radical one for duodenal ulcer. It allows a wide removal while retaining a large gastric reservoir.

The clamp* is shown in Figure 1. The serrated and curved section produces the new lesser curvature of the stomach. This curve allows more of the acid-bearing mucosa to be removed than would a straight clamp. The serrations permit solid and deeply set hemostatic sutures to be well placed on the proximal cut edge, while the stomach wall is being held securely and evenly by the clamp, and crushed only slightly. The straight transverse section (b-c, Fig. 3) crushes not at all but because of the pins, holds just as securely and evenly as does the tighter fitting curve. This straight non-crushing bar preserves a healthy stomach wall for the gastrojejunal anastomosis. The anastomosis can be made any width desired,



FIGS. 1 and 2.

* Made by the J. Sklar Manufacturing Co., New York, N. Y.

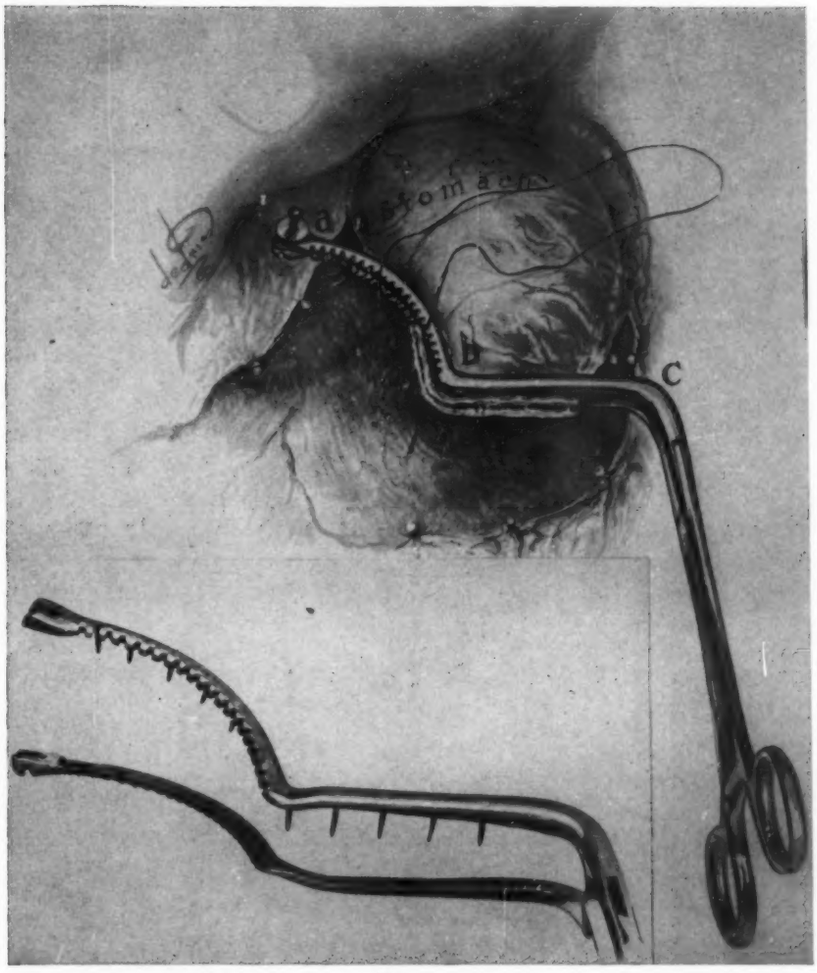


FIG. 3.

up to the length of the crossbar, which is approximately 5 cm. The width of the anastomosis is arranged by applying the handle of the clamp close to or far away from the greater curvature. The farther away, the narrower is the anastomosis. (We prefer a small opening which more nearly approximates the size of the natural pyloric exit of the preoperated stomach.)

The non-crushing qualities and the holding pins can be seen in Figures 2 and 3. The pins are placed along the upper jaw only, so that the jaws do not have to be opened too widely to envelop the stomach wall. The screw-ferrule on the end at (a) is removable so that this end of the clamp may be passed through a hole made in an edematous gastrohepatic omentum above a hard-to-handle high gastric ulcer. Such an application allows the transection to be made with greater ease above the ulcer and, in those cases in which inflammatory reaction has made it difficult or dangerous to interrupt the left gastric artery, with a minimum of blood loss from the cut edge.

Figure 3 shows, diagrammatically, the use of the clamp. After satisfactory devascularization has been accomplished along the curvatures, the clamp is placed as illustrated, and the screw-ferrule is applied and closed tightly. This holds, in a very secure fashion, the high lesser curvature which is sometimes difficult to control. The handle serves as a retractor and a manipulator to control the stomach edge for suturing. In the diagram the stomach wall has been severed about 2 to 3 mm. from the bar and a somewhat smaller fringe has been left along the new lesser curvature. The upper section of the new curvature has been sutured with a continuous No. 000 chromic catgut. This suture is tied near (b) after the clamp has been removed and the sutured fringe has been inspected. If satisfactory, the new lesser curvature is inverted by interrupted Halstead sutures of fine silk. The upper end may be

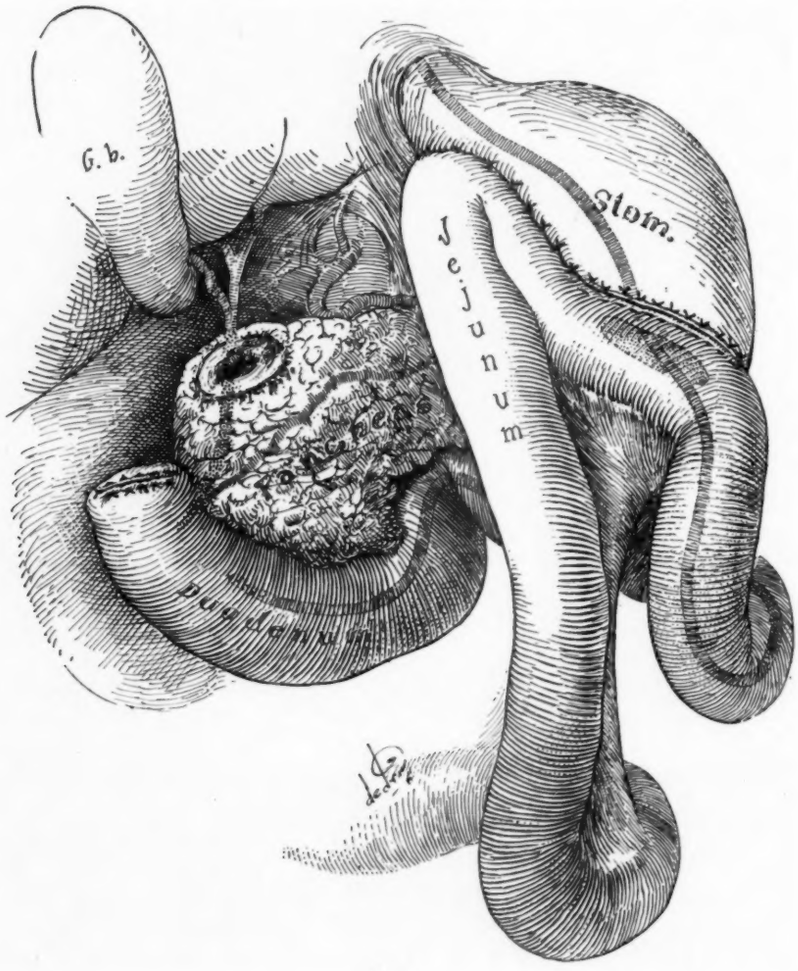


FIG. 4.

buried in an oblong purse string suture and reinforced by the tag of gastrohepatic ligament, previously removed from the high lesser curvature during the latter part of the devascularization process. The inversion is discontinued about two sutures from the angle at (b) so that plenty of lax serosal surface is available for the second row of sutures which complete the anastomosis and for the inversion of the gastrojejunal anastomosis. The stomach tube can easily pass between the jaws of the cross-bar when the clamp is applied to the stomach and this is often a convenience.

In the diagram in Figure 3 the curve on the clamp is shown as being more rounded than it actually is, and the needle is pointed in the wrong direction. A comparison with the clamp as shown in Figure 1 will show that more of the lesser curvature would be removed than is shown in Figure 3. The actual amount removed is better illustrated in Figure 4. This extra removal can also be accomplished if the instrument is moved upward toward the cardia so that the crossbar (b-c) comes closer to the end of the devascularized area on the greater curvature. As small or as large a stomach pouch as is desired can usually be left even though the entire lesser curvature is removed. Although the anastomosis is made in a transverse direction, the finished operation usually appears as in Figure 4,[1] because the weight of the jejunum pulls the greater curvature down a little bit, so that the anastomosis is more oblique than it is straight.

Acknowledgment: The writer would like to thank the Conroy Manufacturing Company, St. Catharines, for making alterations in the clamp from time to time.

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Removable Foam Rubber Surgical Mittens for Retractors, Clamps and Forceps

AN AID IN THE REDUCTION OF RETRACTION AND OTHER SURGICAL TRAUMA

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THE use of removable foam rubber obstetric forceps mittens has succeeded in reducing surface injury to the newborn's head in delivery.[1] The soft tissues of the body actively or passively engaged during operations are similarly in need of protection from the crushing and cutting effect of bare metal.

The application of considerable forces must often be applied to Deaver, right-angled and self-retaining retractors in order to achieve proper exposure. Tears, cuts and ecchymoses of the skin and subcutaneous tissues often occur as a result of these spreading, pulling or pushing forces. Sometimes a loop of intestine gets caught between the retractor and the abdominal wall. The edge of the retractor has been known on occasion to cut the intestine. Bruising of the intestine without actual cutting is more common. Covering the retractors with removable foam rubber mittens, as is so successfully done with obstetrical forceps in delivery, would reduce retraction trauma. (Figs. 1 and 2.)

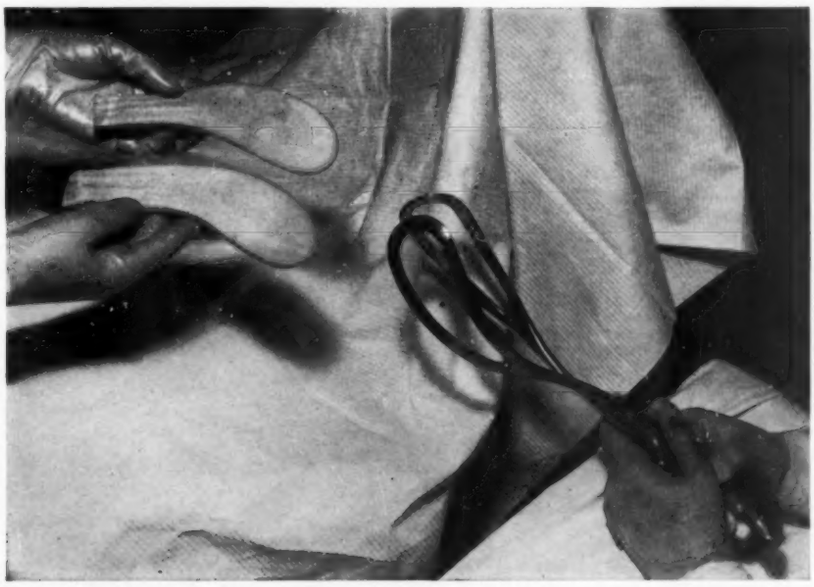

FIG. 1. Removable foam rubber obstetric forceps mittens. When these mittens are placed over the bare steel forceps prior to delivery, the delicate tissues of the baby's head are spared cuts, bruises and ecchymoses.

Sponge forceps (ring clamps), Allis clamps and similar instruments are often used to grasp a viscus or other delicate tissue. It is suggested that removable foam rubber mittens be applied over the bare steel of these forceps to make the latter less traumatic.

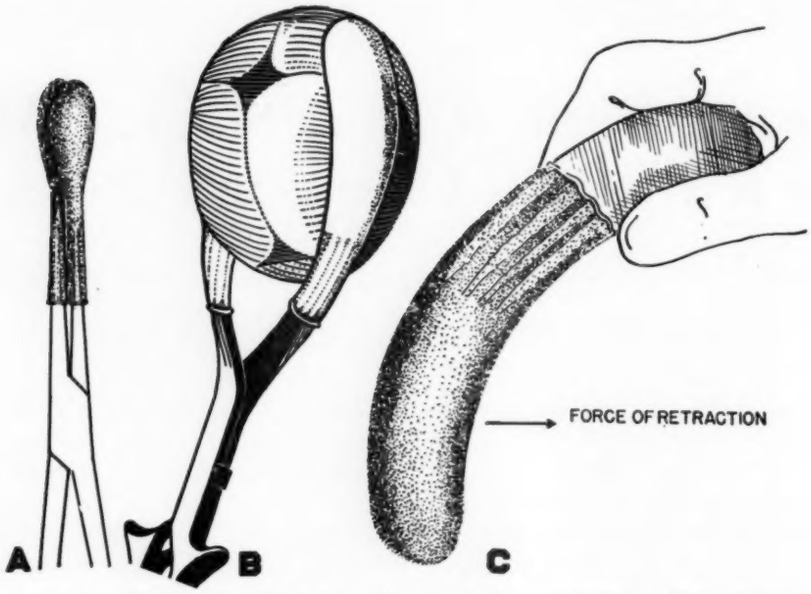

FIG. 2. Examples of use of foam rubber coverings on surgical instruments to prevent unnecessary visceral trauma. A, ring clamps covered with foam rubber mittens; B, obstetric forceps covered with foam rubber mittens; C, Deaver retractor covered with foam rubber mittens.

SUMMARY

Removable foam rubber surgical mittens* for retractors, clamps and forceps are suggested for the relief of retraction trauma in general surgery. As with the use of obstetrical forceps, surgical forceps trauma is similarly reducible with the use of the removable mittens.

* U. S. distributor: The Bittner Corporation, New York, N. Y. Worldwide distributor: The Philips Corporation, Einthoven, Holland and New York, N. Y.

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BOOK REVIEWS

Cardiovascular Surgery. By Gerald H. Pratt, M.D. Philadelphia, 1954. Lea & Febiger. Cloth binding, 843 pages, with 358 illustrations on 261 figures and 4 color plates. *Price* $15.00.

This is a timely and an important book. The author, with a sound basic training and about twenty years of teaching and active clinical work (in clinics and operating rooms), has an adequate and solid background for this volume. It is well written, without padding, and covers the subject in every respect. It was written primarily for surgeons and students, but physicians will find it an ideal refresher postgraduate course, and instructors will find it an up-to-the-minute aid in undergraduate teaching.

This work brings a summary of accepted or acceptable treatment for cardiovascular lesions. The author is refreshing in his frankness. In the Preface he says, "All new reports are not included. It is accepted that tomorrow there may be a new discovery which makes some dogma of this text obsolete."

Assembled in sections, the first section deals with the new nomenclature; the second section discusses anesthesia, cardiac arrest and resuscitation, blood and fluid balance and the employment of antibiotic drugs; the third part is concerned with operations on the heart. Cardiac physiology, thoracotomy and, also, measures are included for both congenital and acquired heart disease. Accepted and experimental measures are considered, as are the treatment of coronary occlusion, tumors of the heart and lesions of the pericardium. The arterial system, injuries to vessels, therapy for aneurysms and embolisms, grafting and blood vessel banks, and the surgical treatment of hypertension are given adequate consideration. The author reviews the venous system, changes in the therapy of lymphatic obstruction, efforts to divert the circulation, methods of bypassing parts or sections of the heart and blood vessels, and other procedures such as angiography, cardioangiography and cardiac catheterization. Skin grafting, the surgical treatment of portal hypertension and the significance of atomic energy and radioactive isotopes, carotid body tumor, carotid sinus syndrome, and the importance of injuries and occupational hazards in a patient with cardiovascular disease and in the person who has had such a disease rounds out the work. This is a pertinent subject, thoroughly and well covered.

Atlas of Exfoliative Cytology. By George N. Papanicolaou, M.D., PH.D. Cambridge, 1954. Published for the Commonwealth Fund by Harvard University Press. *Price* $18.00.

This is an exceptional and outstanding work. Its appeal will be mainly to pathologists and certain laboratory workers; however, surgeons, gynecologists and even workers in the medical fields would do well to become conversant with more than a superficial knowledge of this method of diagnosis.

The preface reads (in part): "The study of exfoliated cells found in various secretory and excretory fluids of the body has received added impetus in the past three decades . . . A real and urgent need has thus been created for publications providing a detailed description and illustration of the enormous variety of cells desquamating from normal or neoplastic surfaces . . . The present work is a further contribution toward this end."

It is of real value in two broad fields: (1) the physiology and endocrinology of the female sex organs and (2) the diagnosis of certain pathologic conditions, more particularly cancer.

There are thirty-six full pages of colored plates which have been beautifully done. The text is brief. The pages are 10½ by 8 inches. The cover is flexible and the text pages are held by the loose-leaf method so that future additions can be easily made to the present book. This is one of the exceptional scientific works of the year. T.S.W.

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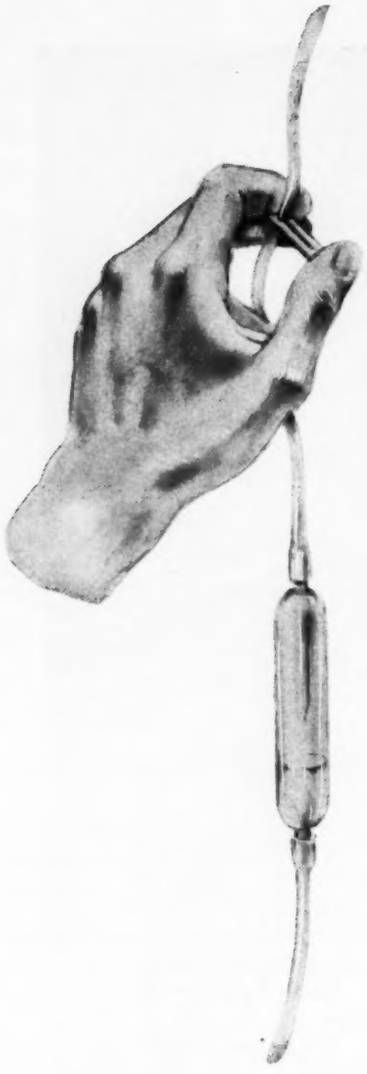

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degree and duration of muscle relaxation

Controllability is the outstanding advantage of 'Anectine'. It is the result of rapid onset of action, followed by rapid inactivation of the drug by hydrolysis.

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'Anectine' *Chloride brand*

SUCCINYLCHOLINE CHLORIDE

Injection . . . 20 mg. in each cc.
Multiple-dose vials of 10 cc.
Ready for intravenous injection.

Solution . . . 50 mg. in each cc.
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To be diluted for preparation of intravenous drip solution.

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Enriched bread, representing the bulk of bread consumed today, makes significant nutrient contributions to the dietary and to the nutritional health of the American people.[1] Bread cannot be regarded merely as an energy food. Instead, it is an important purveyor of many nutrients which a large proportion of our population would never receive in adequate amounts if enriched bread were not available on so large and wide a scale.[2] Here is what modern day enriched bread provides:

VITAMINS: Containing specified amounts of thiamine, riboflavin, and niacin, enriched bread makes a significant contribution to the satisfaction of these vitamin requirements. Enriched bread has played an important role in virtually eliminating frank deficiency diseases and materially reducing subclinical deficiency states resulting from dietary inadequacies in these essentials.[2]

MINERALS: By providing substantial amounts of calcium[3] and of added iron, modern enriched bread goes far in satisfying the needs for these substances. For example, six ounces of bread on the average provides approximately 15 per cent of the day's recommended calcium allowance for adults and 38 per cent of the iron allowance.

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ECONOMY: At its present day low price, bread represents an outstanding nutritional "buy." It provides not only generous amounts of essential nutrients, but also readily available food energy. These features truly make enriched bread one of America's *basic foods.*

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

REFERENCES

1. Sebrell, W. H., Jr.: Trends and Needs in Nutrition, J.A.M.A. *152*:42 (May 2) 1953.

2. Flour and Bread Enrichment, 1949-50: Prepared by The Committee on Cereals, Food and Nutrition Board, National Research Council, 1950.

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4. Sherman, H. C.: Chemistry of Food and Nutrition, ed. 8. New York, The Macmillan Company, 1952, pp. 212; 597-600; 646.

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For skin diseases in which there is an increased pH of the skin surface and/or a hypersensitivity to alkali, such as: Dermatitis of the hands in Surgeons, occupational Eczema due to primary irritants including soap and alkalis, Nummular Eczema, Bath Dermatitis, Winter Itch, Dry Skin and Seborrheic Dermatitis. *Write for Literature and Samples*

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70 Ship Street, Providence 2, R. I.

1. Grayzel, H. G., Heimer, C. B., and Grayzel, R. W.: New York St. J. M. 53:2233, 1953.
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Levo-Dromoran Tartrate 'Roche'...
a new form of synthetic narcotic...
usually longer acting than
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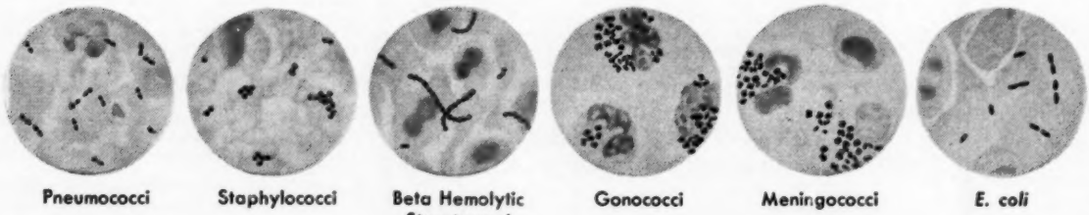

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NOW AVAILABLE:

CAPSULES: 50, 100, 250 mg. • PEDIATRIC DROPS: Cherry Flavored, 10 cc. vials, 100 mg. per cc., Approximately 25 mg. per 5 drops • ORAL SUSPENSION: Cherry Flavored, 1 oz. vials, 250 mg. per teaspoonful (5 cc.) • TABLETS: 50, 100, 250 mg. • SPERSOIDS*: Dispersible Powder, Chocolate Flavored, 12 and 25 dose bottles, 50 mg. per rounded teaspoonful (3 Gm.) • INTRAVENOUS: 100, 250, 500 mg.

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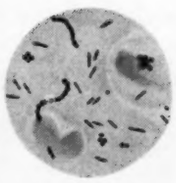

Mixed Infections
(Staphylococci,
H. Streptococci,
Proteus Vulgaris)

Acute Bronchitis
(Pfeiffer's
Bacillus)

NOTES AND QUOTES

LIVING in retirement in Raleigh, North Carolina, is Dr. Hubert A. Royster, a surgeon of national repute, a teacher, a contributor to the literature of his profession over the years and a gentleman in every sense of the word. Recently he sent us a little reprint of his Medical Maxims (*North Carolina M. J.*, July, 1953), with his written notation, "With especial appreciation of all your kindness." We take the liberty to pass on a few of his gems:

"What an antiseptic is a pure life! . . . Sometimes a good forgettery is more useful than a good memory . . . This is the kind of case in which whatever you do is wrong [How true! Ed.] . . . In medicine there is hardly anything new, except what has been long forgotten. Even Hipprocrates wrote a treatise on 'Ancient Medicine.' . . . Some are born sick, some achieve sickness and some have sickness thrust upon them. This is the science of public health in a nutshell. . . . Where one mistake is made by not knowing, ten mistakes are made by not looking, and for ten who look, there is one who observes. . . . Experience may be the best teacher, but some of us are poor pupils. I know a physician who practiced to a very advanced age and was revered for his enormous experience. The man had made the same mistakes every year for sixty years, and everybody called it experience. . . . Always remember the wise words of Montaigne who said, 'When a man is sick and gets well, one cannot say whether his recovery was on account of the remedies used, the lapse of time or his grandmother's prayers.' "

Ernest H. Volwiler, President of Abbott Laboratories, North Chicago, Illinois, and Chairman of the Board of Directors of the American Chemical Society, has been chosen to receive the Chemical Industry Medal for 1954 "for conspicuous services to applied chemistry." Announcement of the award was made by the American Section of the Society of Chemical Industry, donor of the medal. Formal presentation of the medal to Dr. Volwiler will take place at a meeting of the American Section following a dinner in the medalist's honor at the Conrad Hilton Hotel, Chicago, on Tuesday evening, October 12, 1954. The meeting will be held in connection with the National Chemical Exposition.

DEATHS from barbiturate poisoning total somewhat more than 400 yearly in the general population of the United States, or about one and a half times the number immediately prior to World War II, according to the Metropolitan Life Insurance Company's statisticians. Among the life insurance company's industrial policyholders the barbiturates are responsible currently for 30 per cent of all deaths from accidental poisoning. With few exceptions the victims were adults, those in their 30's and 40's contributing a large share of the total, and women outnumbering men in the ratio of two to one.

A variety of drugs and medicines, and such poisons as wood and denatured alcohol, lead, carbon tetrachloride, benzine, kerosene, the caustic alkalis and insecticides account for other deaths from accidental poisoning among industrial policyholders. Almost all the victims of lead and kerosene poisoning were young children. Much can be accomplished in reducing the loss of life, the statisticians point out, by keeping drugs and medicines in properly marked containers, by storing insecticides and disinfectants away from food and other edibles and by keeping poisonous substances out of the reach of young children.

"The barbiturates present a more difficult problem," the statisticians observe. "Greater efforts should be made to acquaint the general public with the dangers inherent in using such drugs carelessly. Further restrictions on the sale of the barbiturates would also tend to reduce the death toll."

The first successful color coding of silk sutures for greater safety in the operating room has just been announced by Gudebrod Bros. Silk Co. The name "Color Coded" has been adopted and copyrighted as identification.

Three colors have been adopted for this use and applied to the three sizes of sutures used in the greatest number of cases. Pink identifies U.S.P. size 4-0, blue 3-0 and white 00. With this distinct color coding, both surgeon and operating room nurse can be absolutely certain the correct size is being used.

With the introduction of Color Coded Silk Sutures, embodying the basic features of the well known Champion Serum-Proof suture line, Gudebrod has made available to the medical pro-

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A new development in the treatment of immobilized patients. While held firmly between two frames, the largest patient can be easily turned by the smallest nurse. Quickly elevated from either end. Easily transportable from hospital to ambulance. Essential for any modern hospital.

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The Primer Bandage (moist) and the Dalzoflex Bandage (elastic adhesive)—suggested in the Pressure Treatment of

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and other ailments of the extremities where pressure is indicated. ***It Costs no more to use the best!***

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fession the principle of color coding in both silk and cotton. Also, both cotton and silk are available in spools and the new Gude-Pack packages of handy pre-cut lengths.

A THIRTY-YEAR account of its fight against diseases of the heart and blood vessels is given in the 1953 Annual Report of the American Heart Association, entitled "The Heart Story." These developments are listed as being of greatest significance since the Association was incorporated in 1924: (1) The availability of more funds for research made possible by the Association's transition in 1948 from a professional organization to a national voluntary health agency, with 56 affiliated heart associations, more than 350 chapters, and Heart Fund campaigns in more than 3,000 communities. (2) Development of rehabilitation programs, clinic services and other community service programs through which newly developed knowledge and technics can be most effectively applied for the benefit of the nation's 10,000,000 cardiac patients. (3) A remarkable change of attitude by both physicians and the general public toward diseases of the heart and blood vessels as a result of a vigorous educational program, with fatalism and fear giving way to a new determination to bring these diseases under control.

The report discloses that in 1953, Heart Fund contributions reached a total of $8,555,197.16, an increase of approximately $2,000,000 over the preceding year, and almost $6,000,000 more than the total received from the first Heart Fund campaign of 1949. "The Heart Story" describes how these funds are spent to advance the Association's comprehensive program of scientific research, lay and professional education, and community services for heart patients.

The report points out that at least 50 per cent of all funds received by the National Office of the Association, are expended to support research projects of outstanding scientific investigators. Out of the total funds raised in the past five years, the Association and its affiliates have devoted more than $7,800,000 to the quest for new knowledge of the heart and blood vessel diseases. Reviewing the great achievements in this field during the past thirty years, "The Heart Story" offers the prospect that intensified and expanded research may make it possible eventually to bring the heart and blood vessel diseases under control.

Among the many community service phases of the Heart Program, a major role is played by the rehabilitation program, the Association's expanding effort to help cardiac patients remain independent, self-supporting members of society. Through a wide variety of activities, the Association seeks to break down the barriers that still keep many capable cardiacs out of jobs. As a service to heart patients, their medical advisers and employers, the Association fosters the development of work classification units or clinics, where the work capacity of the individual cardiac is assessed as a basis for his selective placement in a job which he can perform fully without overtaxing his physical reserves. Programs have also been initiated in many areas for the housewife with heart disease, through which homemakers may learn how to simplify their household routine and conserve their strength.

In the field of rheumatic fever, which strikes primarily at children and young adults, the report calls attention to the large scale campaign launched by the Association during the past year to encourage the widest possible use of the now available preventive measures by physicians and others concerned with the control of this disease. The Association's "Statement on Prevention of Rheumatic Fever," giving detailed recommendations for the use of penicillin or the sulfa drugs by physicians in the management of streptococcal infections that usually precede rheumatic fever, has been widely distributed both through the Association's own scientific journals and other medical publications. High priority also continues to be given to community programs directed toward meeting the current needs of rheumatic fever victims. For a copy of "The Heart Story," write to the American Heart Association, Inc., 44 E. 23rd St., New York 10, N. Y.)

After many months of research and development a fully explosion-proof surgical saw has been announced by the Orthopedic Equipment Company, Bourbon, Indiana. There has always been a great need for an explosion-proof electric bone saw and recently the increased use of more and more volatile gases as anesthetic agents has bolstered this need. When explosions have occurred in the operating room, the results were usually

CONTINUED ON PAGE 48

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Extremely useful in the treatment and prevention of contracted anus (particularly following hemorrhoidectomy), as an aid in perineal dissection and repair following delivery.

Sold only on prescription—at your surgical dealer or ethical pharmacy

Adult Set sizes 1, 1½, 2, 3,—for stretching anal sphincter.
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Infants Flexible Rubber sizes 000, 00, 0—uses above.
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there is a difference as there is—

in our line of German manufactured-Swedish steel instruments that are the *acme of perfection* in design and performance. Made of course, with stainless steel . . . and in spite of the modest price tags, they are an investment in a "*Lifetime of Service.*"

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loss of life or serious injury. The Armed Forces, cognizant of this hazard, have not recently purchased any electrical equipment for operating room use that has not met safety requirements. Likewise, the Canadian Government has not allowed the importation of bone saws in recent months because existing units did not meet safety regulations. Meeting this long-standing need for a safe bone saw and foot switch, this new Bishop surgical saw fully conforms to safety requirements for Class 1, Group C, Hazardous Locations. Both oscillating and rotating, the Bishop surgical saw retains all the advantages of the original Orthopedic electric bone saw, and is now approved by Underwriters Laboratories, Inc. for use in locations containing highly combustible anesthetic agents. All blades, attachments and accessories used with the original Orthopedic electric bone saw are interchangeable and usable on the new explosion-proof unit. The Bishop surgical saw will be made available through surgical supply dealers.

DR. THEODORE G. KLUMPP, President of Winthrop-Stearns Inc., called industry's widely accepted policy of compulsory retirement of workers at the age of sixty-five as "economically wasteful and socially harmful." A man obviously cannot be a capable worker one day and unfit the next day, merely "because he has reached his sixty-fifth birthday," Dr. Klumpp told the Ohio Citizens Council for Health and Welfare recently.

A member of the Hoover Commission's Task Force on Medical Services, Dr. Klumpp stated there are approximately twelve million persons today who are sixty-five years of age or over. In less than thirty years, he said, the U. S. is expected to have at least twenty-four million in that age group, and sixty-six million over forty-five. "Apart from the human consideration of cutting off their means of livelihood and the civic problem caused by an additional tax burden, the age shift in our population promises to become one of the most important factors in influencing the entire economic and political structure of our country. Both government and private industry must realize the error of wasting the contributions of those who have learned to do their jobs well and are able to continue to do so faithfully. Training and experience can only be replaced by training and experience, and when we retire a competent older worker his successor has the burden of supporting him as well as himself. The fact is that some older people are and some are not fit and pulling their weight, and we must devise intelligent methods of determining for all workers which should be kept at their jobs."

Dr. Klumpp called attention to the serious economic problem involved in compulsory retirement of workers. "The national wealth, purchasing power and standard of living in the U. S. rest squarely on productivity, and when large numbers of the population are unemployed they are neither contributing to production, nor are they in position to consume as much. If we did nothing about the growing number of older people we are forcing into idleness, and maintained present day working standards and conditions, we might well be confronted with a potential labor surplus of over thirty million workers by 1980."

We were looking through some reports from The Metropolitan Life Insurance Company. We have heard it rumored that there are more widows [7,500,000] *than widowers in these United States and the Metropolitan confirmed this fact. Is it because the "weaker" sex is truly the stronger of the species? With all their supposed neuroses most women, once convinced they need surgery, readily submit, while the man will hem and haw and seek several doctors hither and yon before he will arrange for hospital accommodations. Maybe, after all, the male is the weaker sex. As one poet put it, "Women are like a willow that bends in every little breeze but breaks not in the tempest." Or, as one cynic pondered, "Do married men live longer or does it just seem longer"?*

THIRD molars transplanted surgically under the protection of sulfa drugs some years ago have developed normally into vital first molars, according to recent reports in *Oral Surgery* and the "Dental Yearbook."

Dr. George J. Collings of the University of Oregon reported normal development in the case of a youth in whose mouth two third molars were transplanted to replace lost first molars. In the operation the transplanted teeth were protected from infection by being sprinkled with sulfanilamide powder. After four years of observation, Dr. Harland Apfel of San

CONTINUED ON PAGE 50

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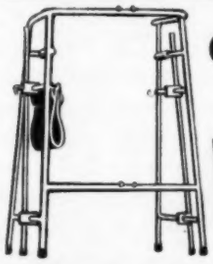

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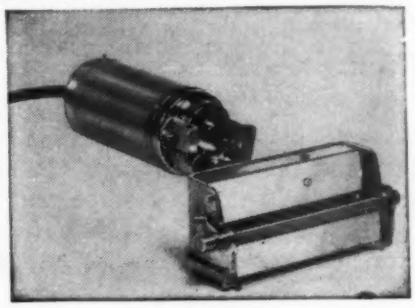

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NOTES AND QUOTES

Pedro, California, also reported success in ten cases in which single third molars had been transplanted to the first molar position in the mouth.

The first molars are the first permanent teeth. Because they determine the alignment of later teeth and affect the shape of the mouth, they are considered the most important of all teeth. More than 100 teenagers have had a lost first permanent molar satisfactorily replaced by one of their own wisdom teeth.

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DECAYED teeth may literally be made as good as new if discoveries announced at an American Chemical Society are confirmed by further research. In the future, cavities may be repaired with materials that allow calcium and phosphate mineral crystals to grow into them, producing fillings indistinguishable from real teeth, Martin Burger of the chemistry department in the Jewish Hospital of Brooklyn told a scientific conference of the Society's New York section. Dr. Albert Edward Sobel, head of the hospital's chemistry department, was co-author of the report.

Mr. Burger and Dr. Sobel believe they have synthesized in the test tube the chemical ingredients responsible for mineral deposition in teeth. "If we are correct, we may be filling teeth in the future by such materials, filling the cavity up in such a way that the new tooth will be at least as good as the original one," Mr. Burger declared, pointing out that these promising new findings need to be confirmed by further studies.

The materials which appear responsible for mineral deposition can now be synthesized by combining collagen (a protein found in bone and skin and widely used in the manufacture of gelatine) with a solid, insoluble substance called chondroitin sulfate, it was reported. The latter is a material whose purpose in the body has never been fully understood, but which in the light of these studies seems to be a chemical promoter similar to the enzymes, key regulators of the processes of life.

A select list of medical films available only to professional audiences may now be obtained free of charge upon request directed to The Princeton Film Center, Inc., Princeton, New Jersey. All films listed are 16 mm., some silent and some sound. Many are in color and include such useful subjects as "Fractures: An Introduction" and "Anomalies of the Bile Ducts." These films are designed for use before student groups as well as by more advanced audiences in the medical profession.

MARCH 24th was the eleventh anniversary of Public Law 16, a Federal law that has enabled nearly two-thirds of a million disabled veterans to train for useful, productive lives, Veterans Administration announced. Its experience in administering this law has demonstrated that veterans with virtually every type of disability can be rehabilitated successfully.

Public Law 16, the Vocational Rehabilitation Act, was enacted March 24, 1943, during the height of the bitterly fought "leap frog" landings in the Pacific and more than a year before the historic D-Day invasion in Europe. Originally designed for disabled veterans of World War II, the law was extended three years ago to cover those disabled in service since Korea. So far 602,000 disabled World War II veterans have trained for jobs they can hold despite their handicaps. Another 13,000 veteran-trainees suffered their disabilities after the outbreak of hostilities in Korea. Today, disabled veterans with almost every conceivable type and degree of disability can be found working in practically every walk of life.

Pharmaceutical and chemical firms awarded twenty-nine grants totaling $95,330 to the University of Illinois College of Medicine during the past twelve months in support of research and

CONTINUED ON PAGE 52

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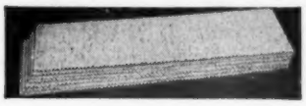

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educational programs. The largest single grant was contributed by Charles Pfizer and Company, Brooklyn, New York, for the continuation of investigations of infectious diseases, preventive medicine and related fields. Dr. Harry F. Dowling of the Department of Medicine is directing this study. Mallinckrodt Chemical Works, St. Louis, Missouri, renewed a $10,000 *grant for a Fellowship in the Department of Pharmacology and for research involving anticonvulsant drugs. Dr. Carl C. Pfeiffer is supervising this research. Eli Lilly and Company, Indianapolis, Indiana, contributed three grants totaling* $9,071. *Abbott Laboratories, North Chicago, Illinois, supported five research projects with grants totaling* $7,100. *Smith, Kline and French Laboratories, Philadelphia, Pennsylvania, awarded two grants in the amount of* $6,000.

THE organic chemical industry sees at least 100,000 new jobs created during the next ten years according to Cary R. Wagner, President of the Synthetic Organic Chemical Manufacturers Association. This expansion in employment is due to the fact that the organic chemical industry is growing at a rate four times faster than all U. S. industry. Speaking before the students and faculty at Wooster College, Dr. Wagner said, "In the best judgment of those of us in the organic chemical industry, we have just begun to grow. Even the most conservative of us talk and think in superlatives—not boastfully, but merely in terms of the foreseeable expansion in employment, plants and the thousands of ways in which chemicals will be used in the future."

The organic chemical industry has become a vital and vigorous part of our national economy, Dr. Wagner said, and its products have become indispensible to all of American industry, to our armed services and to the everyday living of all our citizens. To illustrate his point, Dr. Wagner said that 256 chemicals are used in the manufacture of an automobile. Without chemicals, he added, there would be no steel or lightweight metals, no TV sets or telephones, no synthetic rubber, no sulfa drugs and many other essential products.

"There are no limits to our opportunities," said Dr. Wagner. "For example, it's a good guess that better than half our production of chemicals twenty years from now will be in products unknown to us today."

TWENTY years ago one of five children admitted to the tuberculosis ward of Bellevue Hospital, New York City, died. Today the rate is one of 100. The tremendous saving in lives of children afflicted with tuberculosis is attributed primarily to the use of antituberculosis drugs in treatment in an article by Dr. Edith M. Lincoln in the March Bulletin of the National Tuberculosis Association.

Up to 1946 when streptomycin first became available, children continued to die at the rate of one of every five admitted to the tuberculosis ward, but with the advent of this first successful antituberculosis drug the death rate began to fall and was forced down even lower after the introduction of isoniazid.

In tuberculous meningitis, a severe form of the disease which caused the majority of deaths among the children, the death rate after chemotherapy was first employed fell from 100 to 32 per cent between 1947 and 1951 and, since the advent of isoniazid, has been lowered to about 12 per cent, Dr. Lincoln states.

"The use of antimicrobial therapy in other serious forms of tuberculosis has been even more effective in saving lives," she continues. "Since January 1, 1947, only one child at Bellevue Hospital has died of miliary tuberculosis, which was previously practically 100 per cent fatal, and only one baby died of tuberculous disease of the lung caused by local spread from the primary disease. Thus in one hospital the total death rate from primary tuberculosis and its complications fell from more than 20 per cent to 5 per cent after the introduction of streptomycin, and after the introduction of isoniazid to 1.5 per cent. Therefore, from the standpoint of mortality statistics, chemotherapy has worked wonders in saving the lives of many children with primary tuberculosis."

A newly published booklet, "Reach to Recovery," has been prepared for women who have undergone radical mastectomy. This booklet attempts to help women meet some of the emotional and physical problems with which they are faced following such a traumatic experience. The policy of this booklet is to work through interested hospitals in each community throughout the country. Inquiries for these booklets must come from recognized hospitals or physicians. Individuals or hospitals may write to: Reach to Recovery, 1440 Broadway, New York 18, New York.

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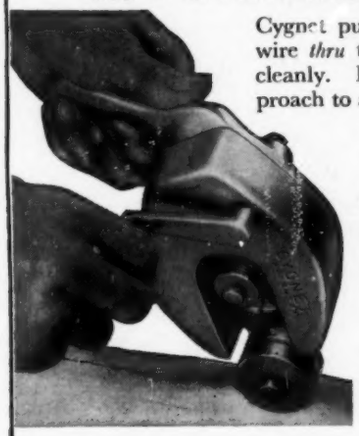

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